Disease Risk Decreases in Diverse Plant Communities Observed along an Elevational Gradient

Master's thesis

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July 2020

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Tiedekunta – Fakultet – Faculty Faculty of Biological and Environmental Sciences		Koulutusohjelma – Utbildningsprogram – Degree Programme Master's Programme in Integrative Plant Sciences
Tekijä – Författare – Author Mikko Jalo		
Työn nimi – Arbetets titel – Title Disease Risk Decreases in Diverse Plant Communities Observed along an Elevational Gradient		
Oppiaine/Opintosuunta – Läroämne/Studieinriktning – Subject/Study track Plant biology		
Työn laji – Arbetets art – Level Master's thesis	Aika – Datum – Month and year July 2020	Sivumäärä – Sidoantal – Number of pages 54 + appendices

Tiivistelmä – Referat – Abstract

As biodiversity is being lost worldwide at an accelerating rate due to anthropogenic activities, the frequency and severity of many infectious diseases has been observed to increase. Together these patterns have brought forth an urgent need to understand the possible linkages between biodiversity and disease risk.

Two contradicting hypotheses have been proposed to explain the diversity-disease relationship. The dilution effect hypothesis suggests that increasing host community species diversity 'dilutes' disease risk, whereas the amplification effect hypothesis predicts disease risk to increase with increasing diversity. Even though most of the studies support the dilution effect, there remains an intensive debate regarding the generality of this effect.

As most of the research efforts to understand the relationship between diversity and disease have focused on animals and crop plants or have been carried out experimentally, one of the research gaps is how relevant the dilution effect is in wild plant communities. In nature, plants and their diseases are affected simultaneously by multiple abiotic and biotic environmental factors that might confound or supersede the effects of diversity. It is also poorly understood, whether we might expect dilution effects to occur not only on diversity gradients driven by anthropogenic diversity loss, but also on natural diversity gradients.

To study the possible association between host community species diversity and disease risk in the wild and to test whether this association could be detected after accounting for the effects of abiotic factors, I surveyed grassland vascular plant communities for their species diversity and foliar disease symptoms along a natural diversity gradient driven by elevation. I also recorded data on the mean soil surface temperature in the surveyed plant communities and used structural equation modelling to differentiate and compare the effects of biotic and abiotic variables on disease risk. The data were collected on Mount Calanda in the Swiss Alps during summer 2019.

In this thesis I show that host community species diversity and disease risk are negatively associated with each other along a natural diversity gradient driven by elevation. Furthermore, this negative effect can be detected even after accounting for the effects of elevation and mean soil surface temperature on disease.

Together the results support the occurrence and the ecological relevance of the dilution effect in wild plant communities along natural diversity gradients and suggest that diversity might protect wild plant communities from increased disease risk. Future studies should aim to identify the exact mechanisms of the association to help us better understand when and where we might expect dilution effects to occur in the wild. This knowledge can be used to predict how epidemics, that affect the well-being of ecosystems, humans and wildlife, are born in the changing world.

Avainsanat – Nyckelord – Keywords
dilution effect, biodiversity, disease risk, plant disease, Alps

Ohjaaja tai ohjaajat – Handledare – Supervisor or supervisors
Anna-Liisa Laine and Fletcher Halliday

Säilytyspaikka – Förvaringsställe – Where deposited
HELDA - Helsingin yliopiston digitaalinen arkisto / HELDA - Helsingfors universitets digitala publikationsarkiv /HELDA - Digital Repository of the University of Helsinki

Muita tietoja – Övriga uppgifter – Additional information

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Tiedekunta – Fakultet – Faculty Bio- ja ympäristötieteellinen tiedekunta		Koulutusohjelma – Utbildningsprogram – Degree Programme Kasvitieteen maisteriohjelma
Tekijä – Författare – Author Mikko Jalo		
Työn nimi – Arbetets titel – Title Kasvien riski sairastua pienenee monimuotoisissa kasviyhteisöissä		
Oppiaine/Opintosuunta – Läroämne/Studieinriktning – Subject/Study track Kasvibiologia		
Työn laji – Arbetets art – Level Maisterintutkielma	Aika – Datum – Month and year Heinäkuu 2020	Sivumäärä – Sidoantal – Number of pages 54 + liitteet

Tiivistelmä – Referat – Abstract

Luonnon monimuotoisuus vähenee kiihtyvällä tahdilla ihmistoiminnan vuoksi. Samalla monien tartuntatautien on havaittu lisääntyneen. Tautiekologian keskeiseksi tutkimuskysymykseksi onkin viime vuosina noussut luonnon monimuotoisuuden ja tartuntatautien välisten mahdollisten yhteyksien selvittäminen.

Monimuotoisuuden vaikutuksista tautiriskiin on esitetty kaksi vastakkaista hypoteesia: laimennus- ja lisääntymishypoteesit. Laimennushypoteesin mukaan tautien isäntäyhteisön monimuotoisuus vähentää tautiriskiä. Lisääntymishypoteesi puolestaan ennustaa, että lisääntyvä monimuotoisuus kasvattaa tautiriskiä. Vaikka suurin osa tutkimuksista tukee laimennushypoteesia, on epäselvää, kuinka yleistä laimennushypoteesin toteutuminen on.

Suurin osa isäntäyhteisön monimuotoisuuden ja tautiriskin suhdetta koskevista tutkimuksista tarkastelee eläimiä ja viljelykasveja. Laimennushypoteesin toteutuminen luonnonvaraisissa kasviyhteisöissä tunnetaan sen sijaan huonommin. Luonnossa kasveihin ja niiden taudinaiheuttajiin vaikuttavat samanaikaisesti useat elottomat ja elolliset ympäristötekijät, joiden vaikutukset voivat olla monimuotoisuutta tärkeämpiä tai häiritä monimuotoisuuden vaikutusten havaitsemista. Ei myöskään tiedetä tarkasti, toteutuuko laimennushypoteesi lähinnä ihmisen luomilla vai myös luonnollisilla monimuotoisuusgradienteilla.

Tutkiakseni korreloivatko isäntäyhteisön monimuotoisuus ja tautiriski luonnonvaraisissa kasviyhteisöissä ja voiko niiden välisen korrelaation havaita sen jälkeen, kun muita ympäristötekijöitä on otettu huomioon, kartoitin niityillä kasvavien putkilokasviyhteisöjen lajimonimuotoisuutta ja niiden lehtien tautioireita korkeuserojen aiheuttamalla luonnollisella monimuotoisuusgradientilla. Mittasin myös niittyjen maanpinnan lämpötilaa ja vertailin eri ympäristötekijöiden vaikutusta tautiriskiin SEM-mallinnuksen avulla. Keräsin aineiston Sveitsin Alpeilla, Calanda-vuorella kesällä 2019.

Tutkielmassani osoitan, että kasviyhteisön lajimonimuotoisuus ja tautiriski korreloivat negatiivisesti tutkimissani luonnonvaraisissa kasviyhteisöissä. Tämä negatiivinen yhteys oli merkitsevä myös, kun otettiin huomioon muiden mitattujen ympäristötekijöiden, maanpinnan lämpötilan ja korkeuden, vaikutus.

Tulokset tukevat laimennushypoteesin esiintymistä ja ekologista merkitsevyyttä luonnonvaraisissa kasviyhteisöissä, joiden monimuotoisuus vaihtelee korkeuden mukaan, ja osoittavat, että monimuotoisuus voi suojella kasveja kohonneelta tautiriskiltä. Jatkotutkimusten avulla tulisi pyrkiä osoittamaan havaitun korrelaation mahdollisia mekanismeja. Lisääntyneet havainnot laimennushypoteesista ja sen mekanismeista luonnossa auttavat ymmärtämään paremmin, minkälaisissa olosuhteissa monimuotoisuus vähentää tautiriskiä. Tätä tietoa voidaan käyttää apuna, kun yritetään ennustaa ihmistä ja muuta luontoa uhkaavien epidemioiden syntyä muuttuvassa maailmassa.

Avainsanat – Nyckelord – Keywords
luonnon monimuotoisuus, laimennushypoteesi, tautiriski, kasvitaudit, Alpit

Ohjaaja tai ohjaajat – Handledare – Supervisor or supervisors
Anna-Liisa Laine ja Fletcher Halliday

Säilytyspaikka – Förvaringsställe – Where deposited
HELDA - Helsingin yliopiston digitaalinen arkisto / HELDA - Helsingfors universitets digitala publikationsarkiv /HELDA - Digital Repository of the University of Helsinki

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Cover photo: View from one of the research meadows, Nesselboden in approximately 1400m above the sea level. Photo: Mikko Jalo

1. INTRODUCTION

1.1. Why does diversity matter for disease dynamics?

Biodiversity is known to affect many key ecosystem functions and services and to contribute to the well-being of humans, wildlife and ecosystems (Cardinale et al. 2012). Currently, biodiversity is being lost globally at an accelerating rate due to human activity (Pimm et al. 2014). The current extinction rates are estimated to be 100–1000 times higher than the rate of natural background extinctions (Pimm et al. 2014; Ceballos et al. 2015). This trend has brought forth an urgent need to understand the mechanisms through which biodiversity is contributing to ecosystem functions (Cardinale et al. 2012).

As biodiversity has declined, the frequency and severity of infectious diseases have increased (Keesing et al. 2006; Jones et al. 2008; Keesing et al. 2010; Fisher et al. 2012; Ostfeld & Keesing 2012; Johnson, Preston, Hoverman, & Richgels 2013). Whether there is a general diversity–disease relationship has thus become an important research question during the recent years (Johnson, Ostfeld, et al. 2015). A majority of studies have found a negative association between host community species diversity and disease risk (Civitello et al. 2015). This phenomenon is referred to as the dilution effect (Keesing et al. 2006).

The dilution effect has been described in various groups of organisms, but most work has focused on animals and less is known of plants and their pathogens (Liu et al. 2020). Since the effects of diversity on disease might differ between animals and plants due to, for example, differences in host movement and host density (Haas et al. 2011), special attention should be paid to studying specifically plant pathosystems.

Studying plant infectious diseases is increasingly important, because they pose a global threat for food safety (Savary et al. 2012). Globally, the costs of plant diseases have been estimated to be around 220 billion dollars per year (Agrios 2005). Diseases cause not only monetary losses, but malnutritional losses as well (R. Yáñez-López 2012). Yearly around 10–14 % of the world's crop is lost because of plant diseases (Agrios 2005; Oerke 2006). As the world's population grows, societies face great challenges in trying to fight hunger and to create sustainable cultivation methods (Tomlinson 2013; Benton & Bailey 2019). Since pest control still highly depends on pesticides and breeding, applying insights from ecological and evolutionary research on plant pathogens might help in implementing more sustainable disease management methods and increasing crop yields (Zhan et al. 2014).

Plant infectious diseases threaten also natural ecosystems. As plants are the primary producers in terrestrial ecosystems, shifts in their abundances can affect upper trophic levels (Haddad et al. 2009). Infectious plant diseases are also an increasing cause of plant endangerment and a potential cause of extinction for some plant species (Anderson et al. 2004; Smith et al. 2006; Fisher et al. 2012).

Despite their negative effects, plant pathogens are also a considerable and important part of biodiversity on earth (Windsor 1998). Parasitism is a common life strategy and plant pathogens themselves contribute to ecosystem functioning by, for example, regulating host densities and affecting interspecific competition between host species (Dobson & Crawley 1994; Hudson et al. 2006; Dobson et al. 2009; Bever et al. 2015).

Considering the important role of pathogens in natural and agricultural systems, improving our understanding of how biodiversity affects disease dynamics may help us to predict how ecosystem processes may be altered in the changing world. This knowledge can be used to benefit both human well-being and the protection of natural ecosystems.

1.2. Plant pathogens and their hosts

Plant pathogens are microbes that can enter plant tissues or cells (infection) and cause a malfunction (disease) (Casadevall & Pirofski 2002). Pathogens causing plant infectious diseases are a diverse group of fungi, bacteria, viruses and nematodes with various life strategies. They grow on the inside or on the surface of plant tissues and can be either intra- or intercellular (grow in plant apoplast or plant symplast respectively) (Jones 2006). Pathogens feed on plant metabolites or, in the case of viruses, use plant cells to reproduce. The disease symptoms caused by pathogens can be, for example, fungal mycelia growing in or on the surface of plant tissues, chlorosis (degradation of chlorophyll) or necrosis (cell death) but the symptoms vary vastly. In general, diseases cause a malfunction that decreases host fitness, which can be detected as, for example, increased host mortality, decreased growth or decreased reproduction (Agrios 2005).

The ability of a pathogen to infect its host is called infectivity, which is determined by the pathogen's genotype. During their evolution, pathogens have adapted to infect one (specialists) or several host species (generalists). Pathogens can spread within the same host individual or between hosts independently via air or water or with the help of a vector. For example, many fungal spores are able to disperse freely in air and land on their hosts, while plant viruses are often dependent on vectors that transmit the pathogen via wounds. Herbivorous sap-sucking arthropods, such as aphids

(Aphidae), are the most important vectors for plant viruses. Some pathogens can also spread vertically from the mother plant to its offspring (Agrios 2005).

Pathogens are divided to be dependent on either host density or host frequency. Pathogens that spread with disease propagules that disperse via air or water are usually density-dependent because their passive way of transmission is more efficient when the host individuals are abundant and close to each other. Frequency-dependent pathogens are usually transmitted by vectors that actively seek and feed on possible host individuals. For frequency-dependent pathogens, host frequency is more important than density, because the vectors find the hosts even in low densities (Burdon & Chilvers 1982).

Plants are immune to most of the pathogens in their environment, but every plant is a host to some pathogens (Agrios 2005). Host competence is determined as the host's capability to get infected (susceptibility), maintain the pathogen (recovery) and spread the disease further to other individuals (infectiousness) (Gervasi et al. 2015). Plant species and genotypes of the same species vary in their competence. Highly competent hosts are highly susceptible, maintain the pathogens well and spread the disease efficiently. Species with low competence have contrary features. Together, all the species occurring in a community determine the community's overall competence, which depends on the competence of each individual species (Agrios 2005).

Plants defend themselves against pathogens with an immune system that operates before and during infection. For example, the cuticula layer on plant epidermis and lignified structures, such as cork, mechanically prevent pathogens from entering the plant (Agrios 2005). When a pathogen enters the plant, plant cells recognize its pathogenic molecular patterns or the pathogenic molecules it produces in the plant (Jones 2006). This recognition induces a resistant response that either kills the pathogen, neutralizes its toxins or prevents the toxins or the pathogen from spreading within the plant. These defense mechanisms can operate locally at the site of infection or systemically in the whole plant (Agrios 2005).

1.3. The role of abiotic and biotic environment in host-pathogen interactions

Diseases have traditionally been studied in single pathogen – single host -systems. This has produced a lot of valuable information on how pathogens infect their hosts, how hosts defend themselves against pathogens and what are the disease symptoms and fitness-costs caused by each pathogen (Burdon et al. 2006). In nature, however, pathosystems consist not only of multiple pathogens and their hosts but also of the typically highly variable biotic and abiotic environment where pathogens and hosts appear (Figure 1) (McNew 1960).

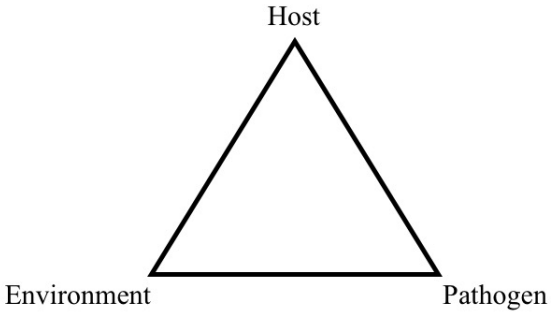


Figure 1. The disease triangle is a plant pathological concept developed to demonstrate how the outcome of the interaction between a pathogen and a host depends on the infective pathogen, susceptible host and the environment. The triangle shows, that infections and/or epidemics occur only when these three factors act together in favor of the disease (McNew 1960).

The important role of pathogens and hosts in disease dynamics is self-evident and thus intensively studied, whereas the effects of the environment on diseases are less known (Warren & Mordecai 2010; Moore & Borer 2012). Abiotic and biotic environmental factors affect both pathogen infectivity and host competence and an optimal environment for an infection to occur is a sum of multiple environmental factors (Burdon et al. 2006; Scholthof 2007). To be able to predict when and where infections and epidemics occur requires expanding the studied system from single host and pathogen species to a broader ecological community context that includes the biotic and abiotic environment of multiple pathogens and hosts (Mitchell & Power 2006; Johnson, De Roode, et al. 2015; Borer et al. 2016; Halliday et al. 2019).

In this thesis, I chose to study the effects of three different environmental factors on disease: host community species diversity (biotic), temperature (abiotic) and elevation (abiotic).

1.4. Relationship between host community species diversity and disease

Biodiversity broadly means the diversity among living organisms. It can be examined on three levels: genetic, population/species and community/ecosystem diversity (Redford & Richter 1999). This thesis focuses on species diversity in a host community which can be measured as 1) host community species richness, measured as the number of host species, 2) host community species evenness, measured as the evenness of which host species are represented in the host community or 3) host community species composition, measured as the abundance the specific entities of host species (i.e. host community species composition) (Redford & Richter 1999; Ostfeld & Keesing 2012).

Host community species diversity has been shown to be one of the environmental factors affecting the risk of an infection (Cardinale et al. 2012; Halliday & Rohr 2019; Magnusson et al. 2020) and two contradicting hypotheses have been introduced to explain the direction of this relationship: the dilution effect hypothesis and the amplification effect hypothesis (Keesing et al. 2006). The dilution effect hypothesis predicts that as host species diversity increases, disease risk decreases. The amplification effect hypothesis, in turn, predicts a positive relationship between diversity and disease.

The idea of diversity 'diluting' disease has a long history (Vandermeer 1989) even though it has been scientifically studied only since the 1940's. Farmers have known for centuries that crop loss to pathogens and pests is lower in polycultures (multiple cultivated species or cultivars) than in monocultures (only one cultivated species or cultivar) (Reiss & Drinkwater 2018). One of the first studies dealing with dilution effect was done in 1958 when diseases were observed to be more abundant in plant communities altered by humans than in natural plant populations (Elton 1958). Already before that, researchers had started to study the diversity-disease relationship with vector-borne human diseases such as malaria and Lyme disease (Brumpt 1944; Service 1991; Matuschka & Spielman 1992). These studies found that as the number of less- or incompetent host species, such as various wild and domesticated animals, increased, the risk of humans to get infected with Lyme disease decreased. These studies were used to develop a broader theory called the dilution effect which was first introduced in 1999 by Norman and colleagues. Since then, the hypothesis has been studied intensively and a negative relationship between diversity and disease has been shown to occur more frequently than a positive relationship (Keesing et al. 2006; Ostfeld & Keesing 2012; Civitello et al. 2015).

Several mechanisms have been identified to cause the dilution effect for both specialist and generalist pathogens and for both density- and frequency-dependent pathogens (Keesing et al. 2006; Ostfeld & Keesing 2012). One of the most important mechanisms is that increases in host community species diversity can decrease host densities. This is because increase in the number of species tends increase interspecific competition between host species (Mitchell et al. 2002; Mitchell et al. 2003). When the susceptible host density is low, the probability of a pathogen to encounter its host decreases (Burdon & Chilvers 1982). This mechanism is particularly important for decreasing the amount of infections caused by specialist density-dependent pathogens that rely on one single host species (Mitchell et al. 2002; Laine 2004; Rottstock et al. 2014).

Dilution effects are also often caused by decreased host community competence in diverse communities. As community diversity increases, highly competent, disease-prone, species may decrease in abundance, whereas they often persist in communities with low species diversity

(Joseph et al. 2013; Johnson, Ostfeld, et al. 2015). The covariance between high resilience and high competence has been attributed to both pathogen adaptation and host life history trade-offs (Ostfeld & Keesing 2000; Joseph et al. 2013). Hosts that persist as communities disassemble or those that recolonize quickly after biodiversity is lost may be poorly-defended against pathogens (Johnson et al. 2012). As species diversity increases, these fast-growing and rapidly reproducing species are replaced by well-defended and long-lived species that tolerate competition (Joseph et al. 2013; Johnson, Ostfeld, et al. 2015). Another explanation for this pattern is that there is an evolutionary bias for pathogens to adapt to infect hosts that are widespread and persist in species-poor communities (Ostfeld & Keesing 2000; Keesing et al. 2006).

Decreased host community competence is an important factor causing dilution effects for generalist pathogens. Even if a pathogen is able to infect multiple hosts, the hosts usually show interspecific variance in their competence, with some hosts species being more competent than others (Haas et al. 2011). As the most competent hosts tend to decrease in density in diverse communities, generalist pathogen abundance may decrease (Keesing et al. 2006; Ostfeld & Keesing 2012).

In the case of frequency-dependent pathogens that are transmitted by vectors, dilution effects in diverse plant communities have been shown to result from decreases in vector abundance and herbivory that suppress disease transmission (Borer et al. 2012; Pagán et al. 2012; Kostenko et al. 2017). A dilution effect may also result if the pathogen is a host-specialist but its vector is a generalist in terms of its nutrition. In such case, the probability of the vector feeding on the pathogen's host plant decreases as plant diversity increases (Keesing et al. 2006).

In addition, disease transmission might be prevented in diverse communities, because non-host species act as physical barriers between host individuals preventing disease propagule dispersal and vector movement (Rottstock et al. 2014; Wäschke et al. 2014; Kostenko et al. 2017). Increased competition in diverse communities might also increase infected host mortality if infected hosts are poorer competitors. As infected host individuals act as disease transmitters, the increase in their mortality can suppress disease transmission (Keesing et al. 2006).

It is also important to bear in mind that despite the fact that many studies have found pathogen species richness to increase with plant species richness due to increased host availability (Rottstock et al. 2014; Dassen et al. 2017; Halliday et al. 2017), pathogen species richness and disease risk faced by the host community are not necessarily positively associated with each other. On the contrary, increase in pathogen species diversity might increase competition between pathogen species and result in decreased pathogen abundances (Harris et al. 2009; Johnson & Hoverman 2012). Therefore, dilution effect might occur even when pathogen diversity increases with host diversity (Rottstock et al. 2014).

Despite the broad evidence for dilution effect, some studies have found host diversity to amplify disease risk (Randolph & Dobson 2012; Young et al. 2013). Mechanisms leading to an amplification effect are contrary to the mechanisms causing dilution effect (Keesing et al. 2006). Diversity increases disease risk when the proportion of highly competent hosts is higher in more diverse communities (Mitchell et al. 2002; Power & Mitchell 2004; Parker et al. 2015; Halliday et al. 2017). Because frequency-dependent pathogens are more reliant on host community composition than host density, they are more likely to experience amplification effects than dilution effects (Halliday et al. 2017). Increased plant diversity might, for example, supply more nutrition for the vectors of frequency-depended pathogens, leading to an increase in vector abundance, herbivory and disease transmission (Keesing et al. 2006). Changes in microclimatic conditions may also underlie an amplification effect. In more diverse communities, greater plant biomass might result, for example, in more moist conditions optimal for pathogen growth (Biggs 1988; Boudreau 1992; Rottstock et al. 2014; Nguyen et al. 2016).

Due to the contradicting findings of the relationship between diversity and disease, researchers' ability to predict when an where we might expect diversity to dilute disease remains poor (Ostfeld & Keesing 2012; Johnson, Ostfeld, et al. 2015; Liu et al. 2020; Rohr et al. 2020). This has fueled an ongoing and lively discussion regarding the strength and direction of the diversity-disease relationship and as well as on the generality and mechanisms of the dilution effect (Randolph & Dobson 2012; Ostfeld 2013; Rohr et al. 2020).

1.5. Relationship between temperature and disease

Another important environmental factor affecting disease is temperature (Truscott & Gilligan 2003). Temperature has been shown to be one of the principal factors to control for species abundances and distributions especially in alpine regions (Laiolo et al. 2018), and hence it is also included as a measured variable in this thesis. As in the case of diversity, temperature may affect both host competence and pathogen infectivity (Paull et al. 2012).

Plants vary in their responses to environmental temperature as species have adapted to different temperatures, but, in general, warm temperatures are favorable for plant growth and reproduction (Colwell & Lees 2000; Allen et al. 2002; Evans et al. 2005; Hoyle et al. 2013). However, the benefits of warm environmental temperatures for plants is not linear and extreme heat might be a source of stress and decrease plant resistance against pathogens, making them more susceptible to infections (Sanden & Moore 1978; Gerechter-Amitai et al. 1984; Roderick et al. 2000; Peng et al. 2004).

Like plant species, pathogen species also differ in their temperature adaptations and the effect of temperature on them is not linear (Roelfs 1992). In general, many pathogens have been shown to benefit from warm environmental temperatures due to increases in pathogen growth and spore germination rate (Tapsoba & Wilson 1997; Harvell et al. 2002; Waugh et al. 2003; Garrett et al. 2006; Avenot et al. 2017). Warmer temperatures can also increase pathogen abundance by increasing pathogen overwintering success (Burdon & Elmqvist 1996; Pfender & Vollmer 1999) or by allowing pathogens to produce more generations during the longer growing season (Garrett et al. 2006). For example, in an experiment in the Rocky mountains, plants that grew on heated research plots showed increased amount of disease damage (Roy et al. 2004).

Even though many studies have shown increasing temperature to benefit pathogens, a negative relationship between disease and temperature has also been observed and the effects of temperature on disease vary not only between pathogen species but also between different life stages and genotypes (Dyck 1983; Wilson et al. 1991; Roelfs 1992; Roderick et al. 2000; Harvell et al. 2002; Araújo et al. 2019). It is also important to bear in mind that extreme heat usually has a negative effect on pathogen growth and survival (Scherm & Van Bruggen 1994; Bourgeois et al. 2004) and that infections occur in an environment that is an optimal combination of several abiotic variables, not only temperature (Agrios 2005). Since many fungal pathogens are dependent on moist conditions and rising temperature decreases humidity, warmer environment might decrease infections caused by fungal pathogens (Agrios 2005; Garrett et al. 2006; Wakelin et al. 2018).

All in all, due to the non-linear and variable responses of hosts and pathogens to changes in temperature, the effects of temperature on plant community disease risk are challenging to predict (Paull et al. 2012). However, several studies have shown that greater pathogen abundances and more severe disease outbreaks are expected to occur in regions with longer growing seasons and favorable pathogen living conditions resulting from warmer temperatures (Harvell et al. 2002; Roy et al. 2004; Garrett et al. 2006).

1.6. Elevational gradients in temperature and species diversity

Elevational gradients on mountains have enabled researchers to unravel many ecological questions (McCain & Grytnes 2010). Elevation creates abiotic and biotic differences over short distances, thereby generating opportunities to study the drivers and patterns of for example species diversity and ecosystem functions (Parmesan 2006; Sanders & Rahbek 2012).

Elevation correlates predictably with many abiotic variables, temperature being one of the most obvious ones. Temperature decreases approximately 0.6 °C per each 100m increase in

elevation because of lower air pressure in high elevations (Barry 2008). This is called the altitudinal temperature lapse rate. The relationship between elevation and temperature has been shown to be linear and the lapse rate to vary from -0.4 °C/100m to -0.7 °C/100m (Barry 2008). The lapse rates vary also within years and within days, usually being lower in winters and during nights. Typical altitudinal temperature lapse rates in the Alps vary from -0.54 °C/100m to -0.58 °C/100m (Rolland 2003).

Species diversity has also been shown to decrease with elevation in alpine regions (Terborgh 1977; Lomolino 2001). This is due to the lower temperature and productivity in high elevations (Odland & Birks 1999). However, although a linear negative relationship between diversity and elevation is a longstanding dogma, in reality, multiple different types of elevation-diversity relationships can occur (Rahbek 1995; Rahbek 2005; McCain & Grytnes 2010). For example, studies have found that elevation can increase species diversity (Dorji et al. 2014) and that the relationship can sometimes be hump-shaped with highest species diversity occurring in mid-elevations (McCain & Grytnes 2010). The shape of the elevation-diversity relationship depends on the studied ecosystem and taxa (Krömer et al. 2013; Peters et al. 2016; Laiolo et al. 2018). For plants, the most frequently observed elevation-diversity relationship is a hump-shaped curve (Rahbek 2005). The peak of diversity in mid-elevations has been attributed to overlapping species distributions (the mid-domain effect) and favorable climatic conditions in mid-elevations (Colwell & Hurtt 1994; Rahbek 1995; Colwell & Lees 2000; Jetz & Rahbek 2001).

1.7. Objectives and hypotheses of this study

Even though most of the previous studies on the relationship between plant diversity and disease support the dilution effect hypothesis (Cardinale et al. 2012; Civitello et al. 2015; Johnson, Ostfeld, et al. 2015), there remains a polarizing debate regarding the generality of this effect (Rohr et al. 2020). This is partly due to the lack of consistent evidence for dilution effect in wild plant communities (Halliday et al. 2020; Liu et al. 2020).

Most of the studies that have detected dilution effects in plants have been experimental (Liu et al. 2020), which limits their ability to prove the ecological relevance of the phenomenon (Ostfeld et al. 2005; Borer et al. 2010; Sagarin & Pauchard 2010; Johnson, Ostfeld, et al. 2015). Experiments are usually undertaken at local scales and controlled conditions (Borer et al. 2010), whereas in their natural environment, plants and their pathogens are simultaneously affected by multiple environmental factors with some of them potentially superseding or confounding the effect of host diversity (Liu et al. 2020).

Artificial communities are also usually built in a way that their species diversity is random with respect to species composition (Joseph et al. 2013). Therefore, experiments include a risk of a sampling effect: diverse communities might include less competent species by chance alone, which leads to a dilution effect (Hector et al. 2002; Joseph et al. 2013). In nature, communities do not assemble at random and host competence is often paired with host resilience (Joseph et al. 2013).

The existing field studies have mainly looked at the diversity-disease relationship on diversity gradients created by human activities and hence, it is not clear how differences in species diversity along natural diversity gradients affect disease risk (Halliday et al. 2020; Liu et al. 2020). The changes in host community composition following anthropogenic disturbance are often predictable and include increase in community competence (Johnson, Preston, Hoverman, & Richgels 2013; Joseph et al. 2013; Johnson, Ostfeld, et al. 2015). However, such predictable changes are not necessarily expected along natural diversity gradients created by abiotic factors, because the effects of abiotic factors on diversity and community competence are variable (Halliday et al. 2020).

Thus, the main objective of this study was to find out whether a negative association between host community species diversity and host community disease risk can be observed in the field in ecologically realistic species assemblages along a natural species diversity gradient driven by elevation.

In particular, this thesis aims to answer the following two questions:

1. Does disease risk decrease with increasing host community species diversity observed along an elevation gradient, as predicted by the dilution effect hypothesis?
2. Are host community species diversity and disease risk associated with each other after accounting for temperature and elevation?

In order to study these questions, I surveyed vascular plant communities for their species diversity and community disease load along a natural biodiversity gradient driven by elevation in the Swiss Alps. I also recorded data of the mean soil surface temperature in the surveyed communities. Based on previous studies reviewed in the introduction, I developed a conceptual model (Figure 2) and drew the following *a priori* hypotheses (1–6) between different environmental variables and how they affect community disease load.

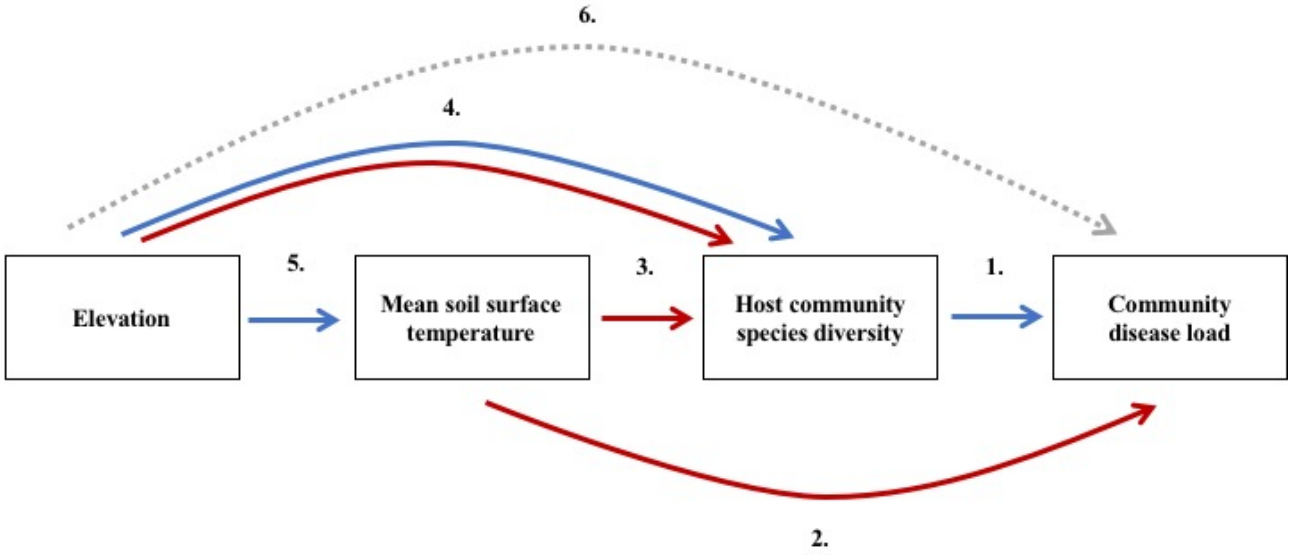


Figure 2. Conceptual model of the hypothesized effects between the four observed environmental variables in this thesis. The effects are presented as arrows with the direction of the arrow indicating the direction of the effect. Blue arrows represent negative and red arrows positive effects. The grey dashed arrow indicates that there is not a significant effect between the two variables. The numbers next to the arrow refer to the hypotheses explained more into detail in the text.

1. Host community species diversity is negatively associated with community disease load.

Multiple mechanisms have been documented to drive the dilution effect in plant communities (Keesing et al. 2006; Keesing et al. 2010; Ostfeld & Keesing 2012; Johnson, Ostfeld, et al. 2015; Liu et al. 2020). I hypothesize, that the same mechanisms operate in nature on a natural biodiversity gradient generated by elevation causing a negative association between host community species diversity and community disease load.

Studies that have found amplification effects have usually been undertaken experimentally with either relatively low species or phylogenetic diversity (Power & Mitchell 2004; Parker et al. 2015; Halliday et al. 2017). In wild plant communities, both species diversity and phylogenetic diversity are usually higher, which might allow dilution effect to occur (Haas et al. 2011). Experiments might also be biased towards amplification effects due to their typically short lengths, because Halliday et al. (2019) show that the effect of diversity might change with time from an amplifying effect to a diluting effect.

It is difficult to hypothesize whether the negative association between host community species diversity can be detected after accounting for mean soil surface temperature and elevation. For example, elevation might decrease both species diversity and mean soil surface temperature. Thus, the increase in community disease load in high elevations caused by low species diversity might

be superseded by the decreasing effect of low mean soil surface temperature. Which of these environmental factors is a more important driver for disease risk depends on the traits of the pathogens and hosts. Therefore I have no *a priori* hypothesis for whether a negative association between diversity and disease can be detected after accounting for the effects of elevation and temperature.

2. Mean soil surface temperature and community disease load are positively associated with each other. This is because higher mean temperatures enhance pathogen growth and reproduction, and therefore increase pathogen abundance (Harvell et al. 2002; Roy et al. 2004; Garrett et al. 2006).

3. Mean soil surface temperature and host community species diversity are positively associated with each other due to more suitable growth conditions in warmer meadows (Tilman et al. 2001; Allen et al. 2002; Evans et al. 2005; Hoyle et al. 2013)

4. Elevation and host community species diversity are either negatively or positively associated with each other (Rahbek 1995).

5. Elevation and mean soil surface temperature are negatively associated with each other, since elevation decreases temperature linearly (Rolland 2003; Barry 2008; Alexander et al. 2015).

6. I hypothesize that elevation *per se* is not directly associated with community disease load, but that it will rather operate by altering other environmental factors such as mean soil surface temperature (Barry 2008; Laiolo et al. 2018).

By using structural equation modeling (Malaeb et al. 2000), this study aims to shed light on the relative importance of multiple simultaneously affecting environmental factors on disease risk with special attention paid for the effects of host community species diversity. The results help to understand what kind of diversity-disease relationships occur on natural biodiversity gradients and to better predict the consequences of the changing environment on the disease dynamics of alpine grassland communities.

2. MATERIALS AND METHODS

To study the association between plant community species diversity and disease risk, I surveyed 220 small plots (d=50cm) along a natural biodiversity gradient for their plant species diversity and foliar disease symptoms and analyzed the data by structural equation modelling. The small plots were established as part of Calanda Biodiversity Project along an elevational gradient (648m–1749m) in Mount Calanda in the Swiss Alps.

2.1. Calanda Biodiversity Project

This thesis was conducted as part of a broader research project called Calanda Biodiversity Project (CBP). The aim of CBP is to study how biotic and abiotic conditions affect plants, their infectious diseases, their pollinators as well as their below-ground microbial communities along an elevational gradient. CBP consists of all publicly owned meadows located below tree-line on mount Calanda in the Swiss Alps. The study setting was established during the first field season in 2019.

For this thesis, I surveyed plant communities for their species diversity, species composition and severity of infectious diseases based on symptom detection in the field to see whether host community species diversity and disease risk are associated with each other and whether this association can be detected after accounting for changes in other environmental variables (elevation and mean soil surface temperature).

2.2. Study area

The study was carried out in the Swiss Eastern internal Alps (Gonseth et al. 2001), on the south-east slope of mount Calanda (46°53′59.5″N 9°28′02.5″E) in the kanton of Graubünden (Figure 3 D). The annual mean temperature in the area at 550m altitude is 10°C and annual mean precipitation is 849mm (Federal Office of Meteorology and Climatology MeteoSwiss 2016) but the climatic conditions vary along the 2804m high mountain. In general, temperature decreases and precipitation increases towards higher altitudes in the area (Barry 2008; Alexander et al. 2015; Federal Office of Meteorology and Climatology MeteoSwiss 2020a; Federal Office of Meteorology and Climatology MeteoSwiss 2020b). In general, the soil in the mountain is calcareous and has low water retention (Eggenberg & Möhl 2007; Alexander et al. 2015).

The mountain is covered by forests surrounding dry open meadows that are kept open by grazing and mowing, a typical form of land use in the Swiss Alps (Bätzing 2015). The study was

carried out on four meadows (Im Bofel, Arella, Nesselboden and Oberberg – Under Alp), publicly owned by the city of Haldenstein (Figures 3 A and 3 C). The meadows are located in different elevations (648m–1749m) with an approximately 500m–1000m distance to each other. The meadows are separated from each other by forests and range from collinean (< 800m) to mountain (800m–1500m) and subalpine (1500–2200m) vegetation zones (Ozenda 1985; Eggenberg & Möhl 2007). The strongest compositional shift in plant communities in the Alps appears between communities located below and above the tree line (Descombes et al. 2017). Thus, in this study, I surveyed only meadows that were located below tree line to control for community composition. The study meadows varied in their size and were grazed by cows two times per year as the cows migrated between low and high altitudes.

2.3. Structure of the study setting

The study setting of CBP consisted of five different units: meadows, sites, large plots, subplots and small plots, nested within each other (Figure 3). Each of the four research *meadows* included 4–7 research *sites*, each with the area of 0.25ha. In total, the project consisted of 22 research sites. Each of the sites contained a grid of nine evenly spaced *large plots* ($4m^2$). The grid with the large plots was placed at random. Each large plot was further divided into four *subplots* ($1m^2$). Five large plots per site contained two *small plots* (d=50 cm) which were placed in opposite subplots and used exclusively for this study. In total, each site had 10 small plots and the whole study setting consisted of 220 small plots.

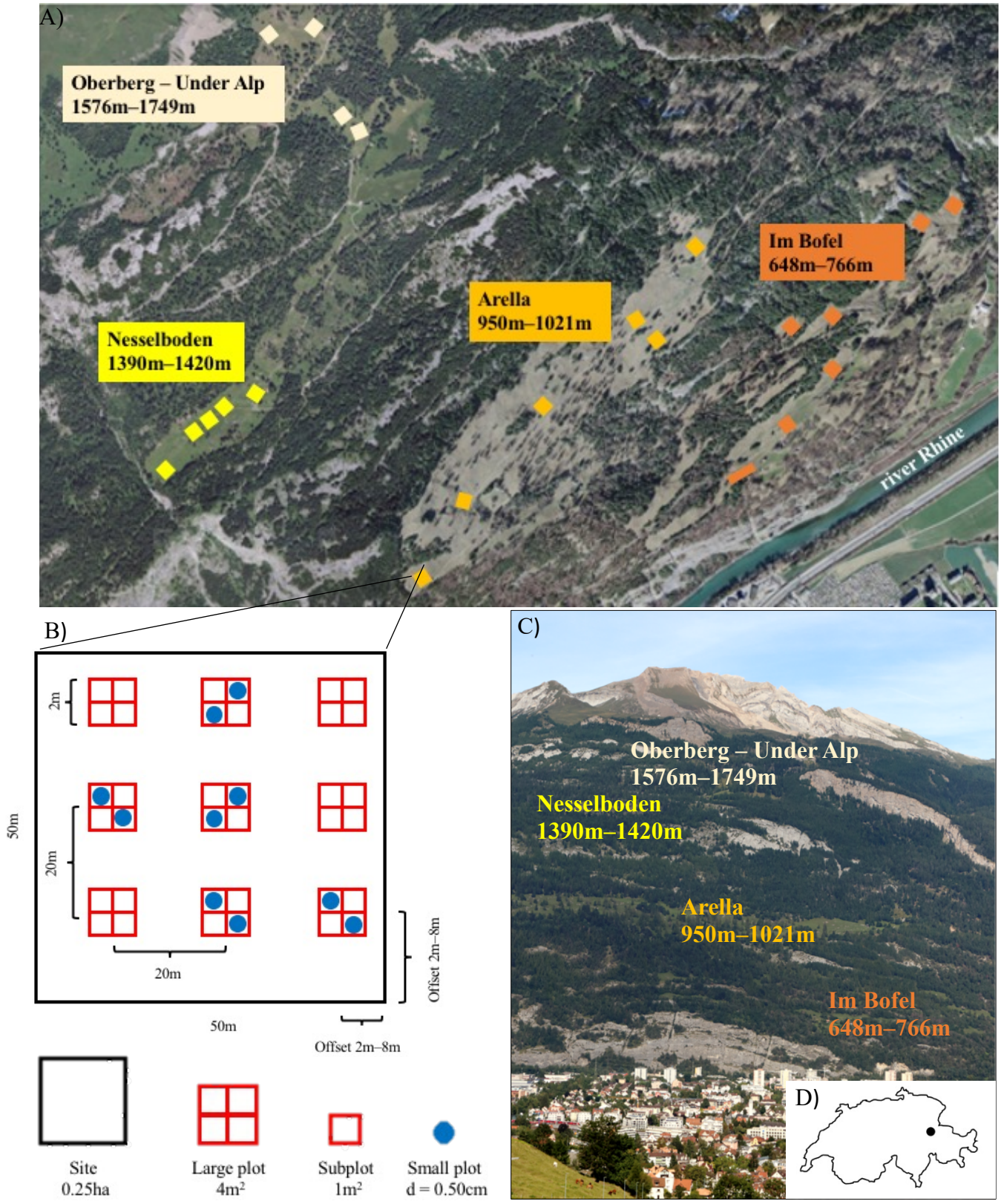


Figure 3. Data sampling design. A) The study meadows and sites on mount Calanda. Photo: Federal Office of Topography SwissTopo 2020, editing: Mikko Jalo B) Example of the arrangement of large and small plots within a site. Illustration: Mikko Jalo C) The study meadows on mount Calanda. Photo and editing: Mikko Jalo D) Mount Calanda marked on a map of Switzerland. Map: Mikko Jalo.

2.4. Establishing sites

For CBP we first established 22 research sites (0.25ha) on the four study meadows (Appendix 1). We fit as many sites on each of the four meadows as possible (4–7 research sites per meadow). When placing the sites, we avoided roads that would cross the sites and large trees, shrubs and rocks that could end up in the middle of the plots creating a forest- or shrub-type habitat that differ from grassland habitats.

The sites were all 50m x 50m except for one site (I3) which was 100m x 25m due to the shape of the meadow. For all sites, one of the corners ('the main corner') was indicated by a visible and stable landmark such as a big stone, tree or stump which we marked with white paint. Other corners were marked with stones that were painted white and buried part-way in the ground. Using a permanent marker, we marked each site identifier (siteID) with the site number on all of the site corners. We also recorded the coordinates of all corners with Swiss Map Mobile application (Federal Office of Topography SwissTopo 2020), and evaluated the approximate elevation of each site based on the coordinates of the main corner.

2.5. Establishing large plots

For all sites, we established nine large plots (in total n=198), each with the area of $4m^2$. The large plots were placed in a 40m x 40m grid within the site with every large plot having a 20m distance to the nearest large plot (Figure 3 B). In order to randomize the location of large plots within the site, the 40m x 40m 'grid' was placed on the site using an offset of 2–8m from haphazardly chosen site corner. We randomized offsets perpendicular and parallel to the river Rhine located next to the mountain (Figure 3 A). The lengths of the offsets were generated with a random number generator and measured in the field with measuring tape.

The center of each large plot was marked with a stone painted white and buried part-way in the ground. Large plots were numbered from 1 to 9 starting from the far-left large plot when the observer was looking at the sites with back facing the river Rhine (Figure 3 B). A plot identifier (PlotID) with site and plot numbers was marked on the central stone with a permanent marker.

All large plots were divided into four $1m^2$ subplots located around the central stone. This was done by placing a $1m^2$ grid on the plot with one of the grid corners on the central stone. The subplots were numbered from 1 to 4 clockwise when the observer was looking at the large plot with back facing the river Rhine.

2.6. Establishing small plots

Small plots (d=50cm) were the main observational unit in this study and they were surveyed for their species diversity and community disease load. For each site, five large plots were chosen to include small plots (Figure 3). Every large plot that included small plots had two small plots that were located in opposite subplots. In total each site contained 10 small plots. Five of these small plots were established based on the density or presence of the plant *Plantago lanceolata* (data collected as part of CBP and reported elsewhere), as this data will be used for another part of the CBP project. The remaining five small plots were selected regardless of the density or presence of *P. lanceolata*.

The first five small plots that were placed based on *P. lanceolata* density, were established as follows. First, one small plot was placed in a subplot having the site maximum *P. lanceolata* density; a second small plot was placed in the subplot with site minimum *P. lanceolata* density; and a third small plot was placed in a subplot with *P. lanceolata* density closest to the median across the entire CBP. These three small plots were always in different large plots. A fourth small plot was placed in a random subplot (by using random number generator) of the central large plot in each site. In the case that the central large plot already had a small plot (n=8), both the large plot and subplot of the fourth small plot were decided randomly using a random number generator. A fifth small plot was always placed at random, using a random number generator to select both the large plot and subplot. Each of these first five small plots were placed in the subplot so that a focal *P. lanceolata* individual was in the center of the small plot. The focal plant was selected haphazardly by placing a hand in the middle of the subplot and choosing the first *P. lanceolata* that touched the hand. We marked the center of each small plot with a grill stick and permanently tagged the focal *P. lanceolata* individual. If the subplot did not contain any *P. lanceolata* individuals (n=6), the small plot was placed in the middle of the subplot.

The first five small plots were placed non-randomly, based on the presence and density of *P. lanceolata*. Previous surveys (results reported elsewhere) of the CBP indicated that *P. lanceolata* density was highly variable among subplots in the same large plot. In order to capture small-scale variation in host community species diversity within large plots, we placed an additional set of five small plots independent of *P. lanceolata* density by placing a small plot in the middle of the subplots located opposite to each of the first five small plots (Figure 3 B). As a result, a site had therefore in total 10 small plots and the whole study consisted of 220 small plots.

2.7. Vegetation survey

I surveyed all 220 small plots for their plant species richness and the coverage of each species using a modified Daubenmire method (Halliday et al. 2019) in the peak of the growing season 27.6.–1.8.2019 together with a field assistant. I started the survey in the lowest elevations and continued higher in order to survey the meadows approximately at the same phase of the growing season in relation to each other. There was a 35-day gap between the surveys of the lowest and highest elevations (Appendix 2). The survey of each meadow was initiated at least four days after cows had grazed the meadows.

In the beginning of the survey on each small plot, a hula hoop with a diameter of 50cm was placed around the central stick to mark the borders of the small plot (Figure 4 A). Plant identification was done at the most accurate possible taxon level with the help of plant identification literature (Eggenberg & Möhl 2007; Eggenberg et al. 2018; Lauber et al. 2018). Plant identification was done either in the field or in the lab with the help of photos and samples together with another surveyor to avoid bias caused by the surveyor (Lepš & Hadincová 1992). Samples were collected only outside the small plots. Due to grazing, all individuals were not possible to identify reliably. If possible, those individuals were marked and identified later in the growing season. An herbarium was collected of all the encountered species and deposited at the University of Zürich.

Coverage of each species was estimated by two surveyors together with the help of a piece of paper that had the cover of 1 % of the small plot to avoid bias caused by the surveyor (Lepš & Hadincová 1992). We estimated how many percentages each species, bare ground, litter, cow feces, rocks, bryophytes and lichens covered from each small plot and summed all coverages to make sure our estimations covered the whole small plot. In order to avoid undermining the coverage of small species, we took into consideration that plants were overlapping. However, the coverages of bare ground, litter, cow feces and rocks were counted only if they were not covered by vegetation. Plant individuals that grew outside the small plot, but had their foliage extend into the small plot, were surveyed as well.

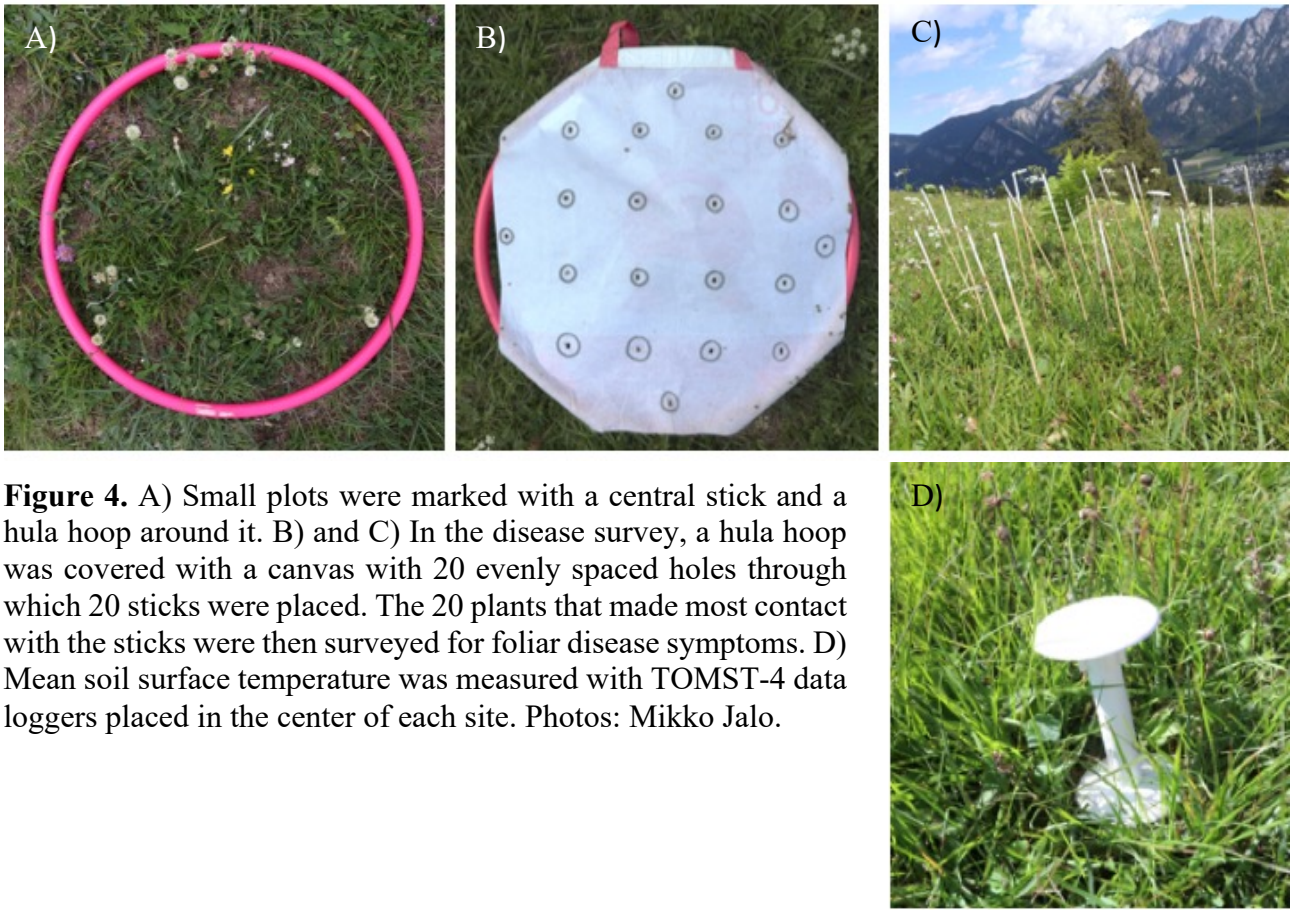


Figure 4. A) Small plots were marked with a central stick and a hula hoop around it. B) and C) In the disease survey, a hula hoop was covered with a canvas with 20 evenly spaced holes through which 20 sticks were placed. The 20 plants that made most contact with the sticks were then surveyed for foliar disease symptoms. D) Mean soil surface temperature was measured with TOMST-4 data loggers placed in the center of each site. Photos: Mikko Jalo.

2.8. Disease survey

I surveyed all 220 small plots (d=50cm) for foliar disease symptoms with the help of an assisting surveyor. Detecting diseases based on symptoms is not as accurate as molecular methods that also detect the presence of asymptomatic pathogens. However, observations of disease symptoms are a better indicator of pathogen abundance and their ecological relevance is higher, since asymptomatic pathogens tend to have lower impacts on plant fitness than symptomatic pathogens (Wilfahrt & Halliday 2020).

Unlike the vegetation survey, the disease survey was not conducted in elevational order due to logistical constrains related to site accessibility. Small plots were surveyed in haphazard order 6–46 days (average 26) after the vegetation survey within a 22-day time period between 29.7.–19.8.2019 (Appendix 2) which we observed to be the peak for pathogen abundance in this system.

In the disease survey, a canvas with a grid of 20 evenly placed holes (with every hole having a distance of 10cm to the nearest hole) was attached to the hula hoop, that marked the borders of the small plots (Figure 4 B). We stuck one grill stick through each hole and lifted the canvas (Figure 4 C). Then, we identified the 20 plant individuals that were most in contact with the sticks and surveyed their five oldest non-senescing leaves for foliar disease symptoms, following the plant

pathogen and invertebrate herbivory protocol (Wilfahrt & Halliday 2020). The survey was carried out on leaves because symptoms are highly visible on leaves. For each leaf we estimated the leaf area (%) that was covered by disease symptoms. We checked both sides of the leaves and excluded dead leaves and tree seedlings from the survey. If the plant individual had less than five leaves, all leaves were surveyed regardless of their age. The observed symptoms were identified and categorized according to Wilfahrt and Halliday 2020. We regularly checked the symptom coverage estimations of the surveyors in order to standardize between surveyors. For each surveyed plant individual, we also recorded species identity.

2.9. Temperature measurements

Temperature was measured in the center of each site (in the center of the central large plot) using a TOMST-4 data logger (Wild et al. 2019) and this single measurement was used in the analysis for all small plots within the same site. In this study I used soil surface temperature which represents the temperature approximately 2cm above the soil surface, where most of the leaves and pathogens were located (Figure 4 D). The thermometers recorded temperature every fifteen minutes for 22–37 days on each site (average 31 days) (Appendix 2). The length of the measurement period varied because some of the thermometers had to be moved earlier or temporarily because of mowing or grazing activities.

2.10. Data analysis

To study the association between host community species diversity and disease risk and whether this association could be detected after accounting for two abiotic variables (elevation and mean soil surface temperature), I fitted two structural equation models (SEM) using the lavaan-package (Rosseel 2012) in R software (version 3.5) (R-Core Team, R Foundation for Statistical Computing, Vienna, Austria, http://www.R- project.org/). SEMs consist of observed variables and hypothetical covariances between them. The benefit of SEMS is that they allow distinguishing both direct and indirect effects and allow teasing apart the effects of confounded variables, such as elevation and temperature (Malaeb et al. 2000). Based on the conceptual model (Figure 2), I fitted two SEMs that included four (1–4) observed variables and the hypothesized effects between them:

1) Host community species diversity

The first model used species richness to quantify host community species diversity and the second model used the effective number of species respectively. This was the only difference between the two models.

Species richness represents the number of observed species in a community (Jost 2006; Chao et al. 2014). The effective number of species in turn takes into account not only the number of species but their evenness as well. Here, the effective number of species means the number of equally abundant species that would be needed to give a particular Shannon's diversity index value (Shannon 1948; Chao et al. 2014). The effective number of species is one of the Hill's numbers (Hill 1973) and is calculated by taking an exponential of the Shannon's diversity index (Shannon 1948) value in a given community as follows:

$$^1D = \exp\left(\sum_{i=1}^{s} p_i \log p_i\right)$$

where ^{1}D = effective number of species, $\sum_{i=1}^{s} p_i \log p_i$ = Shannon's diversity index and p_i = the proportional coverage of the i^{th} species (Chao et al. 2014). For this conversion in R, I used the package hillR (Li et al. 2014).

The advantage of this transformation is that unlike generalized entropies such as Shannon's diversity index, Hill's numbers obey a replication principle, i.e. doubling the number of equally abundant species results in a two-fold effective number of species. This property is useful as it allows to directly compare across different values of effective number of species and, therefore, Hill's numbers are an increasingly used way to measure species diversity (Chao et al. 2014).

2) Community disease load

Community disease load is a broadly used proxy for disease risk in plant disease ecology (e.g. Mitchell et al. 2002, 2003, Liu et al. 2016). I assessed community disease risk in both models by calculating community disease load for each small plot with the following equation (Mitchell et al. 2002)

$$l = \frac{\sum_{i=1}^{n} s_i c_i}{\sum_{i=1}^{n} c_i}$$

where l = community disease load, s_i = disease severity for the i:th species, c_i = coverage for the i:th species. First, I averaged the symptomatic leaf area of all the plant individuals on the same small plot that belonged to the same plant species (disease severity). Then, I weighted disease severity of each species with the relative coverage of that plant species and summed across all species on the small plot. Relative coverage of a plant species was defined as its proportion of the total plant cover. To account for non-normality and heteroscedasticity, I hyperbolic arcsine transformed (asinh) the community disease load data (Halliday et al. 2017).

3) Elevation

I included elevation as a covariate in the model to account for the unmeasured biotic and abiotic variation that is introduced by elevation in these data.

4) Mean soil surface temperature

I included mean soil surface temperature in the analysis as a mean daily average by first calculating the mean temperature for each day and then the mean temperature across all measuring days.

I tested the model fit of both models and accounted for nestedness, non-normality and heteroscedasticity of the data in the models following Halliday et al. (2019). I treated small plots, large plots, sites and meadows as stratified independent grouping variables using lavaan.survey package (Oberski 2014). The meadow Oberberg – Under Alp was split to two meadows (Oberberg and Under Alp) in the analysis due to the within-meadow difference (137m) in elevation.

To study and visualize the bivariate association between community disease load and host community species diversity after accounting for the effect of other variables, I created partial plots of this relationship (Grace et al. 2016).

3. RESULTS

3.1. Vegetation survey

I surveyed all 220 small plots ranging from 648m to 1749m for their species diversity and composition. The total number of observed plant taxa was 189 (Appendix 3). The communities consisted mostly of perennial herbs (for example *Salvia pratensis* and *Helianthemum nummularium*) and were dominated by grasses that tolerate grazing (for example *Dactylis glomerata*, *Lolium perenne* and *Phleum pratense*). The most common species was *Brachypodium pinnatum* (Table 1).

Table 1. Ten most common plant species in the small plots across the whole gradient.

Species	Mean relative coverage
Brachypodium pinnatum	0.183
Bromus sp / Koeleria sp	0.054
Dactylis glomerata	0.044
Unidentified Carex sp 1	0.043
Plantago lanceolata	0.040
Unidentified Carex sp 2	0.037
Phleum pratense	0.034
Lolium perenne	0.033
Carex sempervirens	0.027
Carex flacca	0.022

The species composition of the plant communities changed along the elevational gradient (Figure 5). Nine out of the ten most common species in low (648m–766m) and high elevations (1576m–1749m) were different (Figure 5). Communities in the high elevations (1576m–1749m) located just below the tree line (1800m) and showed features of both low (for example *Lathyrus pratensis*, *Lolium perenne* and *Salvia pratensis*) and high elevation (*Soldanella alpina*, *Ranunculus montanus* and *Carex sempervirens*) species, indicating that the highest surveyed meadows represented an intermediate zone between subalpine and alpine vegetations.

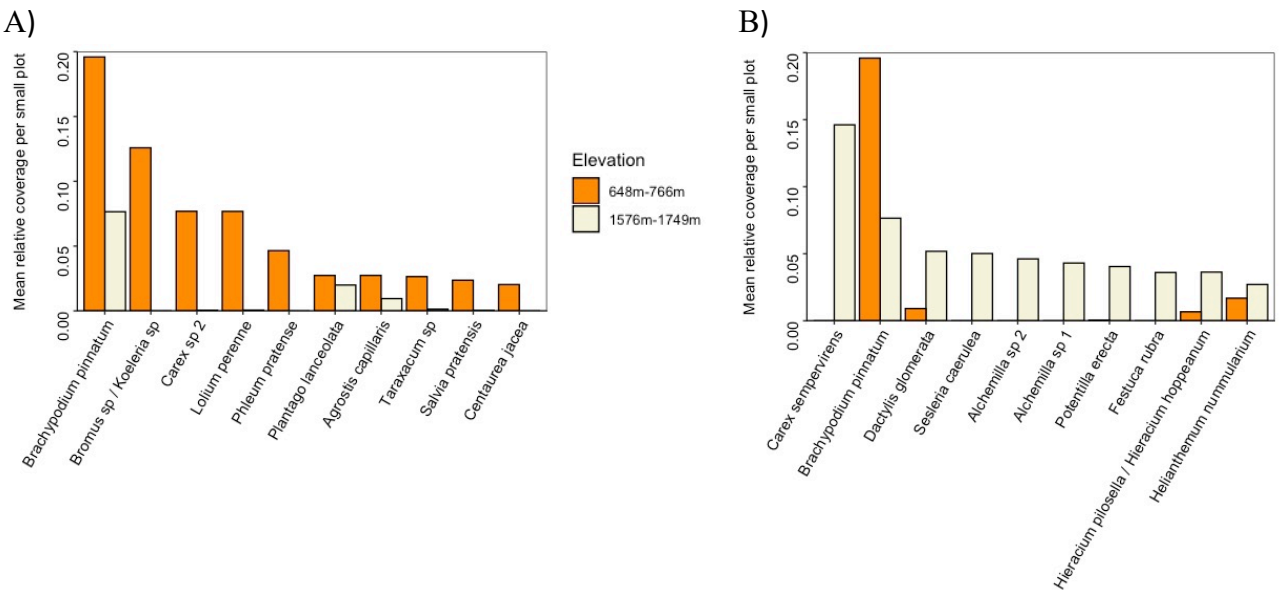


Figure 5. Comparison of the mean relative coverages of the ten most abundant plant species in low and high elevations. A) The ten most abundant species in low elevations (648m–766m) and their mean relative coverage per small plot in low and high (1572m–1749m) elevations. B) The ten most abundant species in high elevations and their mean relative coverage per small plot in low and high elevations.

Small plots showed variation in host community species diversity with both diversity metrics (Figure 6). Species richness varied between 7–30 species per small plot with the average being 20.26 and median 20 species per small plot. The effective number of species varied between 2.74–19.79 effective species per small plot with the average being 9.25 and median 9.15 effective species per small plot. Small plots that were located in the same large plot differed in their effective number of species by an average of 2.31 species.

With both diversity metrics, community species diversity increased with elevation (Figure 6). The median species richness was 16 % and median effective number of species 26 % higher for small plots that were located in high elevations (1576m–1766m) compared to small plots that were located in low elevations (648m–766m) (Figure 6). Together, the results from the vegetation survey showed high variation in host community species diversity along the elevational gradient.

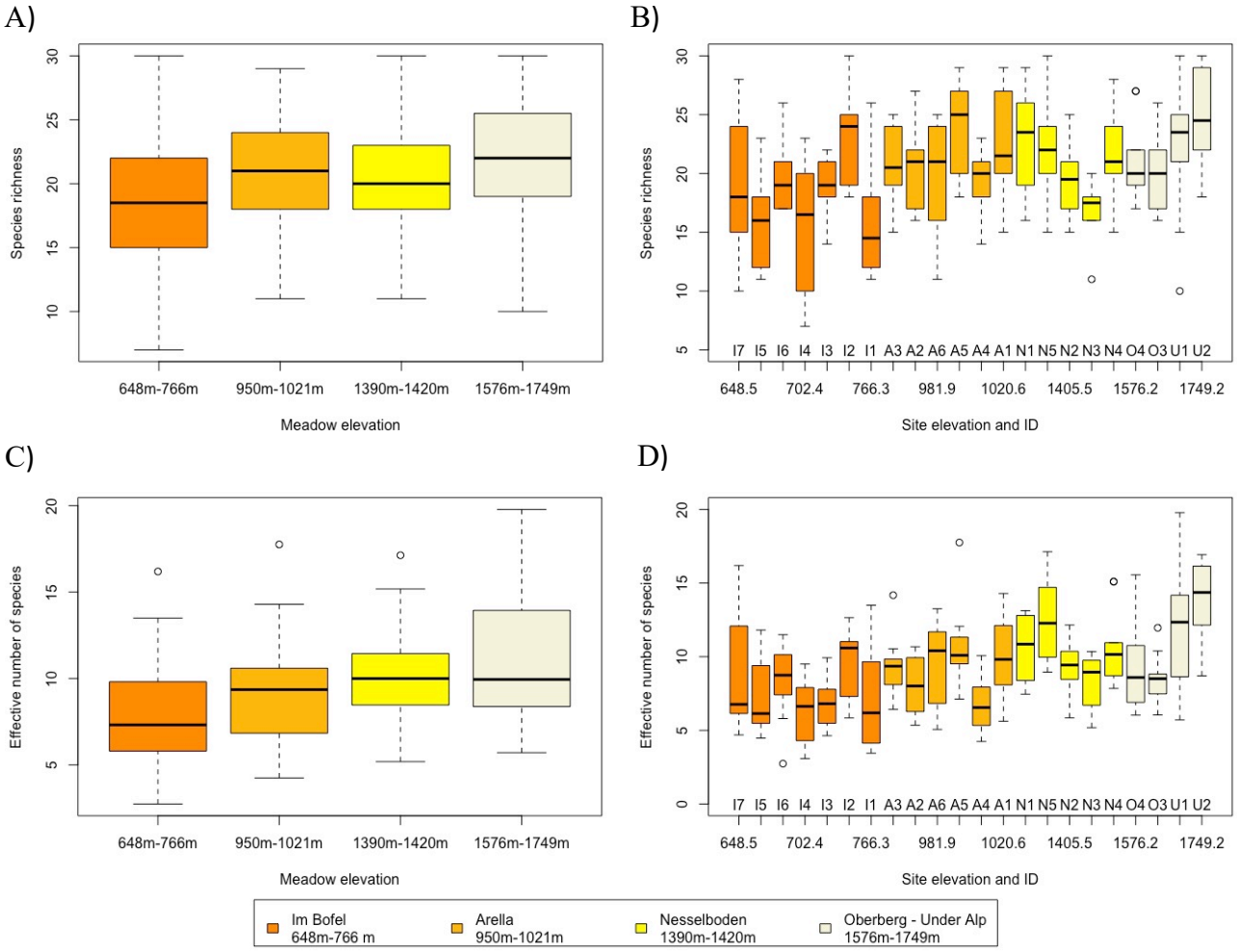


Figure 6. Variation in host community species diversity across meadows and sites. A) The variation of small plot species richness across the four study meadows (represented as different colors) in different elevations (shown in x-axis). B) The variation of small plot species richness across sites with each bar representing a different site and colors representing the four study meadows. Site elevation and ID are shown in the x-axis. C) The variation in effective number of species among meadows. D) The variation in the effective number of species among sites.

3.2. Disease survey

I surveyed all 220 small plots (d=50cm) ranging from 648m to 1749m for visible foliar disease symptoms. In total, I surveyed 18 203 leaves of 4 400 plant individuals belonging to 141 different plant taxa (Appendix 4). The number of surveyed plant taxa was smaller than the number of taxa found in the vegetation survey (189) because the random sampling method excluded some rare species. This has not likely affected the results since the disease severity of each species was weighted by the relative coverage of that species and rare species usually contribute little to community disease load (Mordecai 2011; Heckman et al. 2016).

Disease symptoms were present in all small plots and categorized into six categories (Table 2 and Figure 7). Most of the symptoms were caused by fungal pathogens, such as leaf spot diseases, powdery mildews and rusts. The most common symptom type was leaf spots. This symptom was present on every small plot (Table 2).

Table 2. Occurrence of symptoms on the small plots (n=220). The category 'Other fungal' includes unidentified fungal symptoms. The category 'Other' includes chlorotic and necrotic spots, leaf wetting, leaf curling and leaf choking.

Symptom category	**Proportion of small plots where symptom occurred**
Leaf spot	100 %
Rust	36 %
Blight	38 %
Powdery mildew	16 %
Other fungal	30 %
Other	12 %

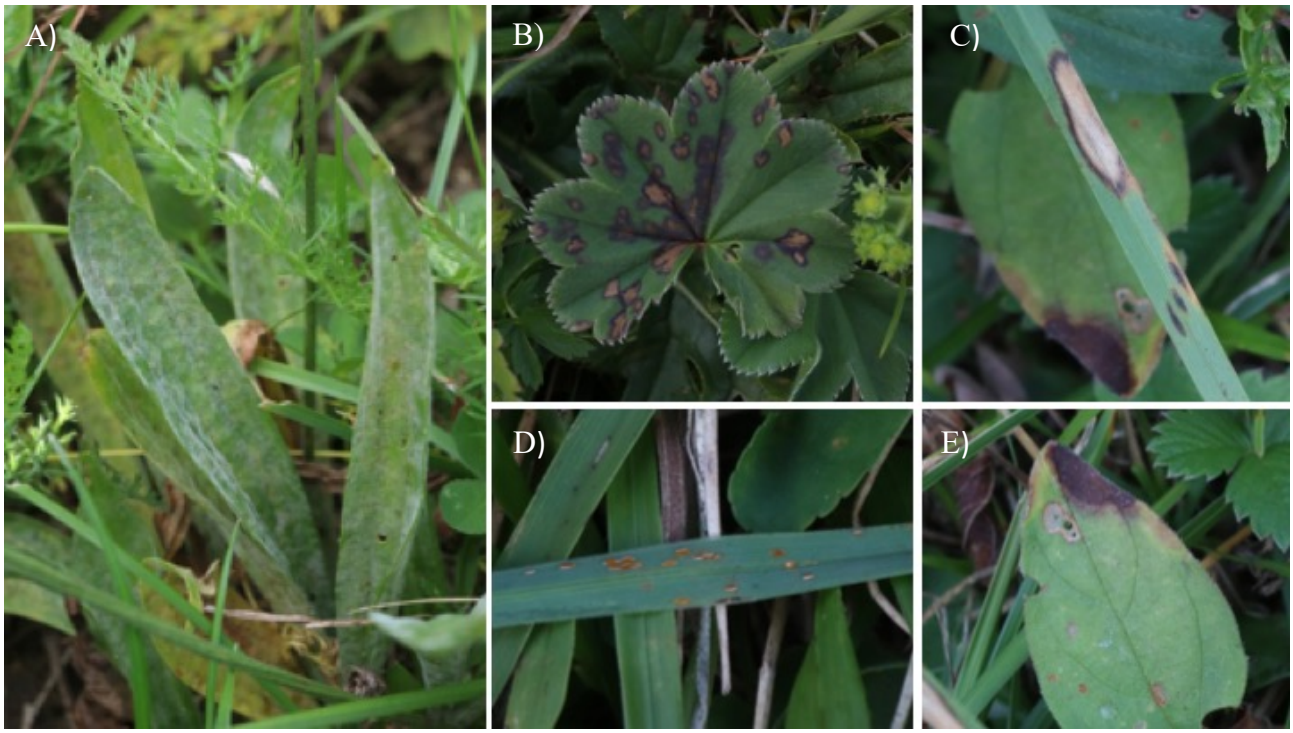


Figure 7. Photos of the observed disease symptoms. A) powdery mildew on *Plantago lancelata*. B) Leaf spots in *Alchemilla sp*. C) Leaf spots on *Dactylis glomerata*. D) Rust on *Sesleria caerulea* E) Blight on *Prunella vulgaris*. Photos: Mikko Jalo.

Community disease load varied across small plots between 0–2.24 with the average being 0.50 and median 0.40. On average, small plots that were located on the same large plot had a difference of 0.36 in their community disease load. Median community disease load per site varied between 0.1–0.79 with the median being 0.26.

Community disease load decreased along the elevational gradient (Figure 8). Median community disease load of small plots in high elevations (1576m–1749m) was 53 % lower than the median community disease load of small plots in low elevations (648m–766m). Together, the results from the disease survey showed high variation in community disease load along the elevational gradient.

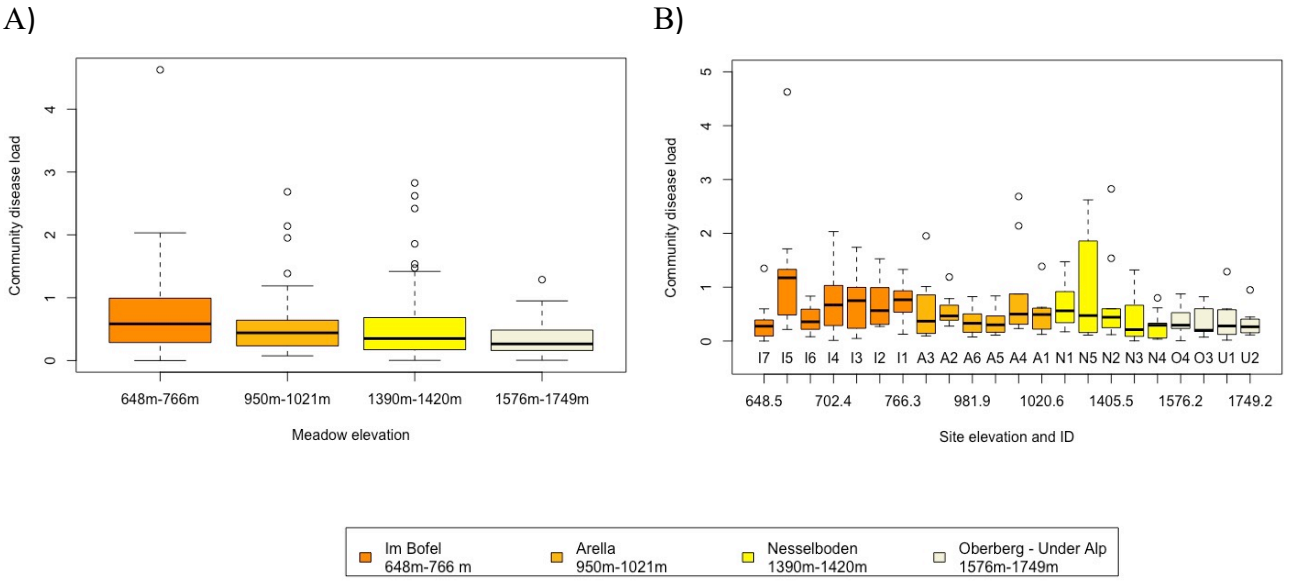


Figure 8. Variation in small plot community disease load across meadows and sites. A) Variation of small plot community disease load across the four study meadows (represented as different colors) located in different elevations (shown in x-axis). B) shows the variation in small plot community disease load across sites with each bar representing one site. Site elevation and ID are shown in the x-axis.

3.3. Temperature measurements

Mean soil surface temperature varied between 13.50 °C–19.72 °C across sites with the average being 16.86 °C and median 16.82°C. Mean soil surface temperature decreased with elevation (Figure 9). Median mean soil surface temperature for sites that located in high elevations (1576m–1749m) was 4.67 °C (26 %) lower compared to sites that located in low elevations (648m–766m). The altitudinal temperature lapse rate along the elevational gradient was -0.57 °C/100m.

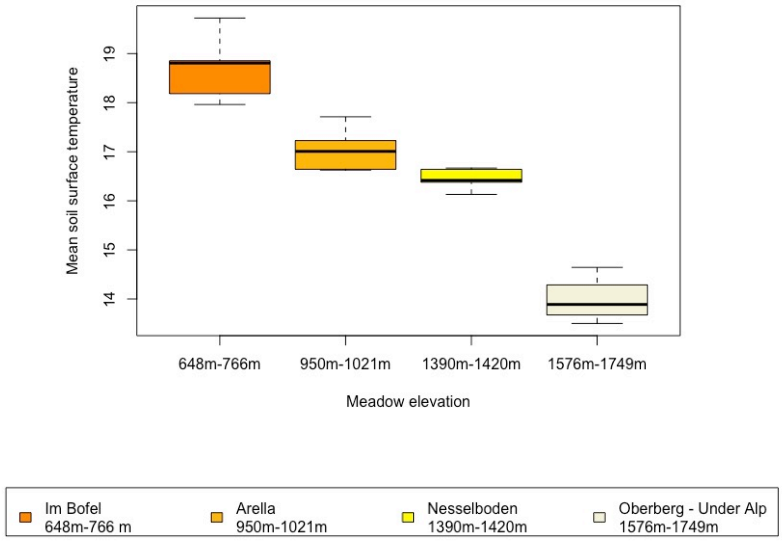


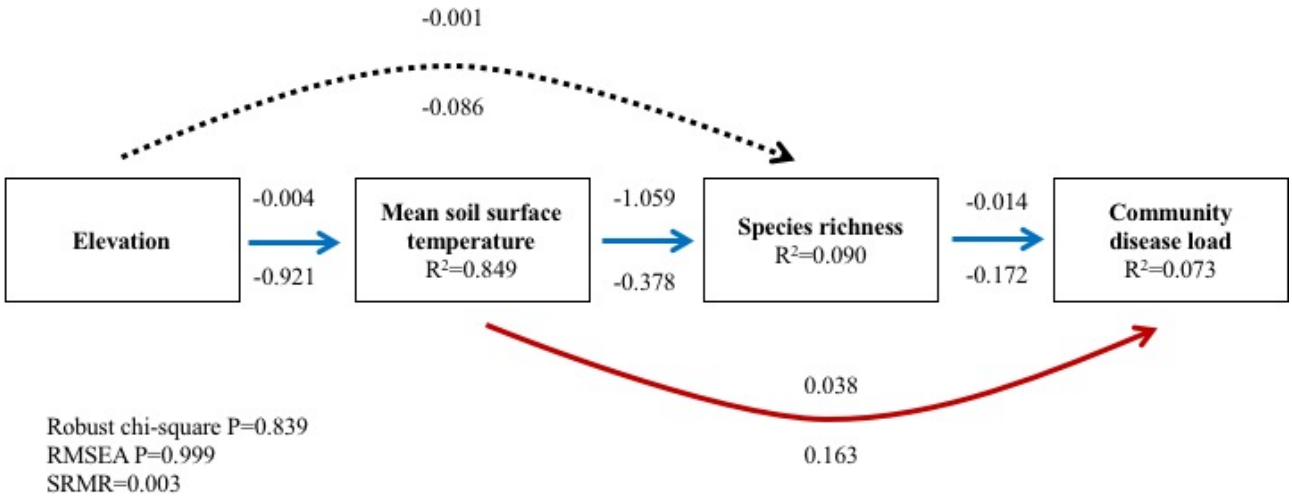
Figure 9. The variation of mean soil surface temperature across the four study meadows.

3.4. Structural equation models

To test the hypothesis that community disease load decreases with increasing host community species diversity and to answer the questions whether this association could be detected after accounting for the effects of other environmental variables (elevation and mean soil surface temperature), I fitted two structural equation models each using a different metric for host community species diversity (model 1: species richness, model 2: effective number of species) (Figure 10).

I hypothesized that elevation *per se* would not affect disease, but rather that elevation would operate through altering other abiotic factors. Consistent with this hypothesis, elevation *per se* did not have a significant direct effect on community disease load in either of the models. Therefore, I removed this non-significant path from the models, adding a degree of freedom in the model to be able to test model fit. The data were well fit by these models (Figure 10).

A)



B)

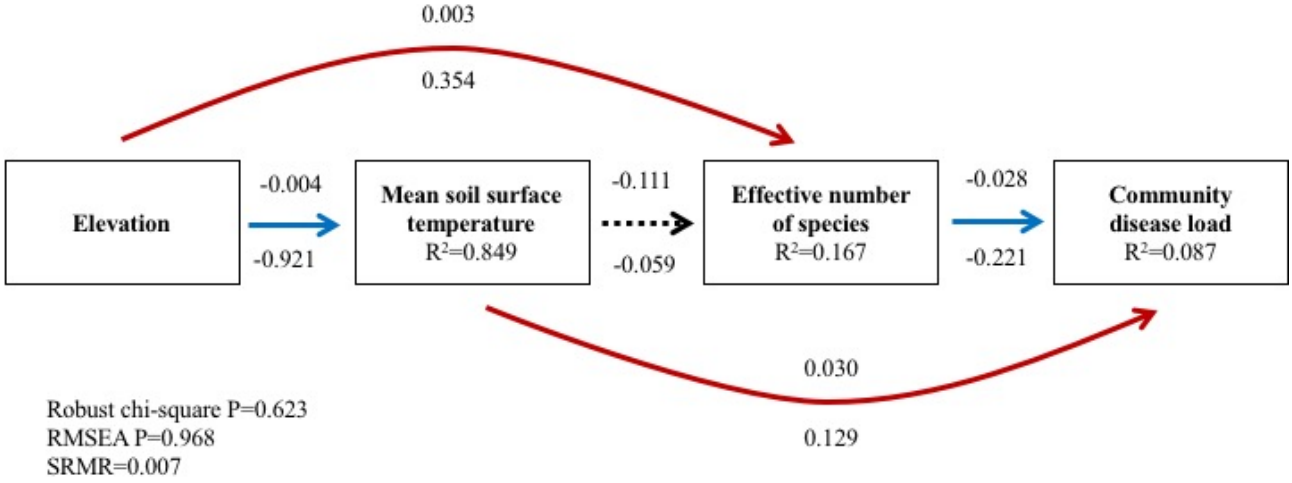


Figure 10. Results from model 1 (A) and model 2 (B). Solid lines represent significant effects ($p<0.05$). Dashed lines represent non-significant effects ($p>0.05$). Blue lines represent negative effects and red lines positive effects. Numbers above the arrows are the unstandardized coefficients between the variables and numbers below the arrows are the within-model standardized coefficients.

Host community species diversity was negatively associated with community disease load in both models so both species richness (magnitude: 0.172, p=0.009) and the effective number of species (magnitude: -0.221, p=0.009) had a negative effect on community disease load (Figures 10 and 11).

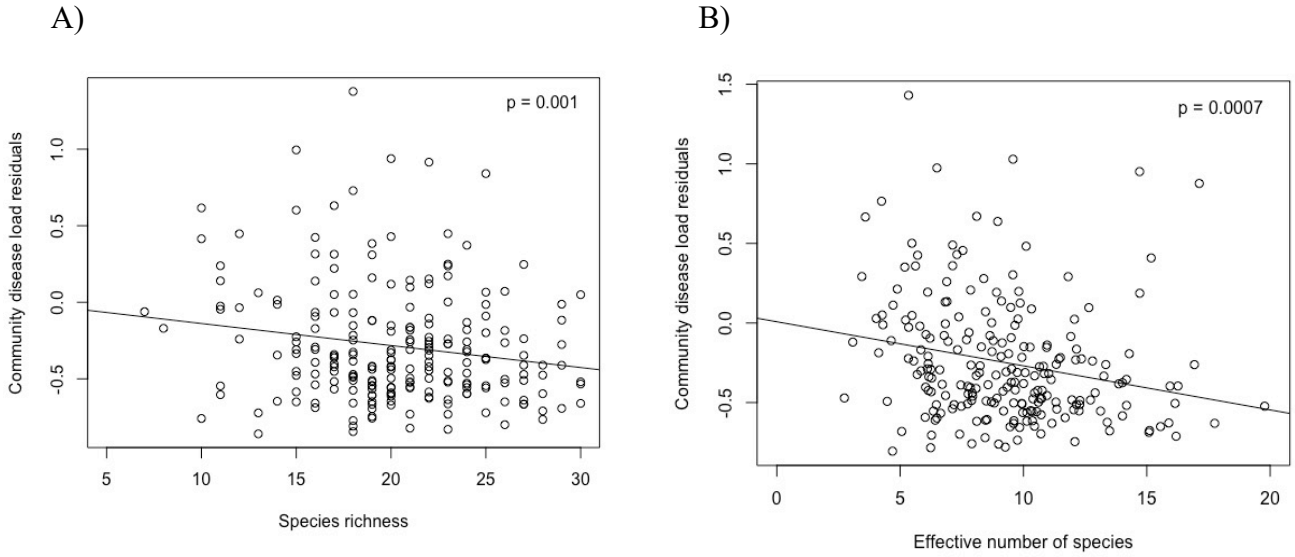


Figure 11. The bivariate association between host community species diversity and community disease load after accounting for the effect of mean soil surface temperature. In partial plot A) host community species diversity is represented as species richness and in plot B) as the effective number of species. Host community species diversity and community disease load were negatively associated with each other with both diversity metrics.

The effective number of species was a better explanatory variable for community disease load (unstandardized coefficient: -0.028) than species richness (unstandardized coefficient: -0.014) (Figure 10). Mean community disease load was 24 % lower in communities with more than 20 species compared to communities with 20 or fewer species. This negative association between diversity and disease existed in both models even after accounting for the effects of all other observed variables on disease.

In both models, community disease load was directly affected not only by host community species diversity but also by mean soil surface temperature (model 1: p=0.006, model 2: p=0.032). This association was weaker (model 1: magnitude 0.163, model 2: 0.129 magnitude), than the association between diversity and disease (model 1: magnitude -0.172, model 2: magnitude -0.221). The effect of mean soil surface temperature on community disease load was positive in both of the models, i.e. small plots with higher mean soil surface temperature had a greater community disease load. Mean soil surface temperature itself was in both models negatively affected by elevation

(magnitude: -0.921, p=0.000). This means, that in both models, elevation had an indirect effect on community disease load via changes in mean soil surface temperature, i.e. small plots that were located higher had lower mean soil surface temperatures which contributed to lower community disease loads.

In addition to influencing community disease load via mean soil surface temperature, elevation also indirectly affected community disease load via host community species diversity. This indirect effect was negative in both of the models. However, the models differed in the structure of this indirect pathway. In model 1 that included host species richness, elevation had a direct negative effect on mean soil surface temperature, which in turn had a direct negative effect on species richness (magnitude: -0.378, p=0.037). In model 1, elevation *per se* did not have a direct effect on species richness whereas in model 2, that included the effective number of species, elevation had a direct positive effect (magnitude: 0.354, p=0.049) on the effective number of species. In model 2 mean soil surface temperature did not have a significant effect on the effective number of species (p=0.741). This suggests, that mean soil surface temperature was the most important factor in the elevational gradient affecting species richness. By contrast, the effect of elevation (or some unmeasured variable associated with elevation) on the effective number of species was more important than the effect of mean soil surface temperature.

Despite the significance of the effects, both models explained only very little of the variation in community disease load (model 1: R^2=0.073, model 2: R^2=0.087) suggesting that also other unmeasured variables were affecting community disease load.

4. Discussion

In the era of increasing loss of biodiversity due to anthropogenic activities, it has become crucial to understand how the diversity among living organisms affects ecosystem functions, such as disease dynamics (Cardinale et al. 2012). For this reason, there has been a tremendous interest to understand the relationship between biodiversity and disease risk (Ostfeld & Keesing 2012; Civitello et al. 2015; Liu et al. 2020; Rohr et al. 2020). Although the diversity-disease relationship has been studied intensively for decades and most of the studies support the dilution effect, there remains a polarizing debate regarding the generality of this effect (Rohr et al. 2020). We especially lack an understanding of how consistently dilution effects occur in wild plant communities along natural diversity gradients.

This observational study shows that disease risk and host community species diversity are negatively associated with each other in the wild plant communities that were surveyed along an elevational gradient in the Swiss Alps on Mount Calanda. Furthermore, the negative effect was detected even after accounting for the effects of two important abiotic variables on disease: elevation and mean soil surface temperature. Together, these results suggest that diversity may dilute disease risk in natural plant communities along the surveyed gradient. In the next chapters, I discuss the main findings of the study and consider how they contribute to our understanding of the dilution effect.

4.1. Host community diversity increased with elevation

In this study, species diversity, measured as species richness and the effective number of species, increased with elevation (see hypothesis 4). The result contradicts the common belief that increasing elevation linearly decreases species diversity (Lomolino 2001). However, in reality the relationship between elevation and species diversity has been shown to vary (Rahbek 1995; Rahbek 2005; McCain & Grytnes 2010; Dorji et al. 2014; Laiolo et al. 2018). For plants, the elevation-diversity relationship is most often hump-shaped, i.e. plant species diversity peaks in mid-elevations and decreases towards both ends of the elevational gradient (Rahbek 1995). This hump-shaped elevation-diversity relationship has been suggested to result from overlapping distributions of both low and high elevation species and optimal growth conditions in mid-elevations (Colwell & Hurtt 1994; Rahbek 1995; Colwell & Lees 2000; Jetz & Rahbek 2001).

Since I surveyed plant communities only up to 1749m on a 2804m high mountain, the highest surveyed meadows represented mid-elevations of a larger elevational gradient and therefore, the observed positive association between elevation and species diversity could exhibit a part of a hump-shaped elevation-diversity curve, with the highest surveyed meadows representing the peak of

species diversity. This is supported by the observation that the highest surveyed meadows included species from both low and high altitudes, which indicates that they were located in an intermediate zone between subalpine and alpine vegetation zones, which could lead in increased species diversity (Colwell & Lees 2000). Nonetheless, for this interpretation to be confirmed, a vegetation survey across the whole elevational gradient would need to be carried out.

Based on the structural equation model, the effect of elevation on species richness can be entirely attributed to mean soil surface temperature, which decreased with elevation as hypothesized (hypothesis 5) (Rolland 2003; Barry 2008; Alexander et al. 2015). This is because after accounting for the effects of mean soil surface temperature on species richness, the effect of elevation became non-significant. The association between mean soil surface temperature and species richness was positive, as hypothesized (hypothesis 3), i.e. warmer small plots showed greater species richness possibly due to favorable growth conditions (Allen et al. 2002; Evans et al. 2005; Hoyle et al. 2013).

Interestingly, the effect of elevation on the effective number of species was not attributed to mean soil surface temperature, because after accounting for the effect of elevation, the effect of mean soil surface temperature became non-significant. This suggests that the effects of some unmeasured variables, that covary with elevation, may have been more important than the effect of mean soil surface temperature on the effective number of species. Such unmeasured variables could be, for example, interspecific competition or facilitation. Interspecific competition decreases and facilitation increases with elevation which might lead in more even species abundances in high elevations (Choler et al. 2001).

It is possible that grazing has affected the results of the vegetation survey (Scott & Hallam 2003). Firstly, herbivory causes defoliation (Trlica & Rittenhouse 1993), which impedes species identification and might be a cause of errors in the data. To avoid misidentification, some observed taxa were grouped into larger taxonomic groups or species pairs (Appendix 3) for which we could be certain of their identity. This, however, might have led to an underestimation of total plant diversity. Secondly, herbivory affects plant size (Pfab & Witkowski 1999), which was used to estimate the relative abundances of each species. If the small plot was grazed recently prior to the vegetation survey, the estimated relative abundances might have differed from the actual relative abundances.

To control for the effects of grazing, all meadows were surveyed at least four days after grazing, but the time between grazing and the survey was not standardized due to logistical constrains and thus some meadows had more time to recover from grazing than others. Also, the duration and intensity of grazing varied between and within meadows. This might have altered species relative abundances since plants show interspecific variation in their grazing tolerance and the amount and

rate of compensatory growth after grazing (Coughenour 1985; Huhta et al. 2003). The rate and pace of recovery from grazing are also affected by environmental factors such as temperature and moisture that differed among meadows (Oesterheld & McNaughton 1991).

In addition to the possible effects of grazing, species diversity may have been affected by the other environmental factors as well. Such variables that were not included in this study are, for example, moisture (Dorji et al. 2014), minimum and maximum temperatures (Vonlanthen et al. 2006) or nutrients (Janssens et al. 1998). The fact that almost half of the small plots were placed based on the presence of *P. lanceolata* is unlike to have biased the results since *P. lanceolata* was a very common species along the gradient and was often present also in plots that were placed randomly. Together, the effects of grazing and other environmental factors that were not included in the models may account for some of the unexplained variation in plant species diversity in the structural equation models.

4.2. Community disease load decreased with host community species diversity

I measured disease risk as community disease load and found that it was negatively associated with both host community species diversity metrics, which is consistent with my hypothesis (hypothesis 1) and the existing literature (Keesing et al. 2006; Keesing et al. 2010; Ostfeld & Keesing 2012; Johnson, Ostfeld, et al. 2015; Liu et al. 2020). In addition, this association remained significant in the structural equation models even after accounting for the effects of elevation and mean soil surface temperature. Together, the results indicate that a dilution effect might operate along the studied natural biodiversity gradient.

The negative association between host community species diversity and community disease load was stronger when diversity was measured as the effective number of species compared to when diversity was measured as species richness. This could be explained by the differences in these two diversity metrics (Magurran 1988). While species richness is purely a measure of the number of species in a given community, the effective number of species represents the number of equally abundant species needed to produce a given Shannon's diversity index value (Hill 1973; Jost 2006; Chao et al. 2014). Shannon's diversity index, in turn, incorporates both the number of species and the relative abundances of species (Shannon 1948). Since many pathogens are sensitive to host density, the effective number of species might affect community disease load more strongly than species richness (Keesing et al. 2006; Johnson, Ostfeld, et al. 2015). When the number of effective species is high, species evenness in the community is high and dominance low. As more abundant

dominant species may be more competent (Arneberg et al. 1998), increase in species evenness might decrease disease (Mitchell et al. 2002).

The fact that a negative association between community disease load and host community species diversity was observed along a natural diversity gradient is interesting since previous studies show that dilution effects are observed more consistently on biodiversity gradients driven by biodiversity loss than on natural biodiversity gradients (Halliday et al. 2020). This is partly because community competence changes more predictably along diversity gradients driven by diversity loss than along diversity gradients driven by abiotic variation (Herms & Mattson 1992; Johnson, Preston, Hoverman, & Richgels 2013; Nobis & Schweingruber 2013; Pellissier et al. 2014; Bruns et al. 2019; Kergunteuil et al. 2019). It may be that in this study community competence decreased with increasing diversity the same way it has been shown to decrease when the diversity gradient is driven by biodiversity loss. Other mechanisms may also underlie the observed association. For example, the encounter rates between pathogens and hosts may decrease in more diverse communities because species with low-competence act as physical barriers between competent hosts (Keesing et al. 2006).

Altogether, this study serves as an example of when we might expect diversity and disease to be negatively associated with each other along a natural diversity gradient. Moreover, it enhances our understanding of the dilution effect by providing an observation of a negative association between diversity and disease in wild plant communities, which are underrepresented in the literature (Liu et al. 2020). Unlike many experimental studies, this study was carried out in ecologically realistic species assemblages and under multiple simultaneously affecting environmental variables, thus gaining more support for the ecological relevance of the dilution effect (Ostfeld et al. 2005; Borer et al. 2010; Sagarin & Pauchard 2010; Johnson, Ostfeld, et al. 2015).

However, since the study was observational, no causal role can be assigned to the observed association between host community species diversity and disease risk. In order to prove that there is a causal relationship between increased diversity and decreased disease, future studies should aim to manipulatively show the mechanisms that may underlie the observed negative association.

4.3. Community disease load increased with increasing temperature

In addition to host community species diversity, community disease load was also positively associated with mean soil surface temperature, as hypothesized (hypothesis 2) (Harvell et al. 2002; Roy et al. 2004; Garrett et al. 2006). Higher disease load in warmer plots could be a consequence of

increased pathogen growth, survival and infectivity and longer growing seasons compared to cooler small plots (Tapsoba & Wilson 1997; Waugh et al. 2003; Roy et al. 2004; Avenot et al. 2017).

Although mean soil surface temperature was positively associated with community disease load, its effect was not as strong as the effect of host community species diversity. This contradicts with previous studies that have shown that in alpine environments abiotic factors such as temperature are usually overriding with respect to biotic factors (Cannone et al. 2007; Laiolo et al. 2018). This study therefore highlights the importance of also biotic factors as possible drivers of disease in alpine environments.

The results also support my original hypothesis that elevation *per se* would not affect disease risk, but that it would rather operate through altering other environmental variables (hypothesis 6) (Barry 2008; Laiolo et al. 2018). In both models, the direct effect of elevation on community disease load was non-significant, but elevation was negatively correlated with mean soil surface temperature and positively correlated with host community species diversity and these variables were, in turn, associated with community disease load. With decreasing mean soil surface temperature and increasing host community species diversity acting together, community disease load was at its lowest in the highest surveyed elevations.

4.4. Other factors that might have affected community disease load

The models explained only very little of the variation in community disease load, which means that most of the variation in community disease load was explained by abiotic or biotic variables that were not included in this study. Due to the fact that elevation did not have a significant direct effect on disease in either of the models, these unmeasured variables appear to be independent of the elevational gradient. This is because if the variables would have covaried with elevation, they should have arisen in the models as a significant direct effect of elevation on disease. On the other hand, it is possible that the unmeasured variables covaried with elevation but had opposite effects on community disease load and therefore canceled each other out, resulting in no net effect.

One of these unmeasured variables could be host community composition that determines the overall host community competence, which affects community disease load (Mitchell et al. 2002; Haas et al. 2011; Johnson, Preston, Hoverman, & Richgels 2013; Joseph et al. 2013). In this study I aimed to control for host community composition by surveying communities below tree-line, because the strongest compositional shift in plant communities takes place below and above tree-line (Descombes et al. 2017). However, I still observed compositional variation within meadows and along the elevational gradient. Several studies have found that increasing elevation selects for

species that are long-lived, slow-growing and well-defended, which could lead to decreased community competence and decreased community disease load in high-elevations (Herms & Mattson 1992; Nobis & Schweingruber 2013). On the other hand, some studies have found opposite trends (Pellissier et al. 2014; Bruns et al. 2019; Kergunteuil et al. 2019) and therefore detailed analyses of plant species traits should be carried out to estimate the effects of host community composition on community disease load along the surveyed elevational gradient.

In addition to host community composition, many other variables might have affected community disease load. Moisture tends to increase with elevation (Frei & Schär 1998; Barry 2008) and it can both enhance and suppress infections and pathogen growth (Biggs 1988; Emery & English 1994; Feil et al. 2003; De Wolf et al. 2003; Laine & Hanski 2006; Warren & Mordecai 2010). UV-radiation may be more intense in high elevations and it can directly kill disease propagules (Manning & v. Tiedemann 1995; Paul & Gwynn-Jones 2003). Also nutrient content may have varied within and between meadows which may affect disease load through altering the amount of foliar nutrients available as resources for pathogens (Huber & Watson 1974; Paul 1990; Jensen & Munk 1997; Nordin et al. 1998; Strengbom et al. 2002; Mitchell et al. 2003).

The results of the disease survey have also likely been affected by mismatches in the timing of the survey between small plots. The vegetation survey was carried out starting from the low elevations to follow the natural proceeding of the growing season, whereas the disease survey was carried out in haphazard order due to logistical constrains related to small plot accessibility during the survey. Small plots that were surveyed later in the growing season might show higher community disease load due to more advanced epidemic: as the epidemic proceeds, more infections and bigger and more visible symptoms accumulate in the plants (Imhoff 1982). Since the timing of the survey was random, it is not likely that the mismatches would have resulted in any kind of trend. On the contrary, they more likely balanced the differences in community disease load between small plots that located in different elevations and weakened the observed indirect association between elevation and community disease load.

Lastly, also grazing and differences in grazing intensity and duration between meadows might account for some of the unexplained variation in community disease load. Grazing can both decrease and increase disease risk (Zhang et al. 2020) through altering microclimatic conditions (Gao et al. 2018), removing pathogens (Skipp & Lambert 1984; Gray & Koch 2004; Wennstrom & Ericson 2018), increasing wounding (Daleo et al. 2009) or altering pathogen-host interactions (Zhang et al. 2020).

All in all, there remains a large variety of environmental variation outside the scope of this study that could affect community disease load. Thus, to be able to explain a larger amount of

the variation in community disease load, future studies should aim to incorporate more environmental factors.

Despite the fact that host community species diversity explained only very little of the variation in community disease load, its effect could still be ecologically important. Previous studies have shown, that even small changes in community disease load might have important effects on host communities and ecosystem processes (Mitchell 2003; Mitchell et al. 2003). Thus, the importance of host community diversity as a driver of community disease load should not be undermined based on the results.

4.5. Conclusions

This study found a negative association between host community species diversity and host community disease load in wild plant communities along a natural diversity gradient driven by elevation. Furthermore, the negative association between host community species diversity and community disease load was detected even after accounting for the effects of two abiotic variables, elevation and mean soil surface temperature. Together, the results support the ecological relevance of the dilution effect in wild plant communities along natural diversity gradients.

While many previous field studies have found dilution effects on single host or single pathogen species (Borer et al. 2010; Seabloom et al. 2010; Haas et al. 2011; Moore & Borer 2012), this study shows that diversity is negatively associated with the whole plant community disease load. Understanding this kind of community-level responses is important as we aim to predict the effects of the changing environment and disease dynamics on ecosystem functions (Mitchell et al. 2003; Mitchell 2003; Johnson, Preston, Hoverman, & La Fonte 2013; Johnson, Preston, Hoverman, & Richgels 2013). Altogether, the results suggest that biodiversity may protect plant communities from increased disease risk, and thus help maintain natural community and ecosystem processes (Combes 1996; Rohr et al. 2020).

This information is increasingly important as alpine grasslands are currently under an increasing pressure by changes in climate change and land use (Theurillat & Guisan 2001). As temperatures increase, plant species diversity has been shown to increase in high elevations due to the upward migration of plant species (Grabherr et al. 1994; Walther et al. 2005; Holzinger et al. 2008; Parolo & Rossi 2008). On the other hand, changes in land use are decreasing grassland species diversity as the traditional ways of agriculture are being replaced with more intensive farming (Maurer et al. 2006; Bätzing 2015). Based on the results of this study, it is possible that the changes in plant diversity in the future may also alter plant disease dynamics.

As the effect of diversity on disease is often context-dependent (Halliday & Rohr 2019; Liu et al. 2020), these results cannot be generalized to other systems (but see Civitello et al. 2015, Magnusson et al. 2020). Our understanding of the circumstances under which dilution effects occur in wild plant pathosystems is still fragmentary (Liu et al. 2020), and to fill these knowledge gaps, future studies should aim to gain more observations of the phenomenon in various wild plant pathosystems. As most of the studies on ecosystem functions have focused on temperate grasslands (Clarke et al. 2017), more observations should arise also from less studied vegetations zones such as tropical and arctic regions. The accumulated observations of the occurrence of the dilution effect help us understand when and where diversity decreases disease risk. This knowledge is crucial, as we aim to predict how epidemics that affect the well-being of ecosystems, humans and wildlife are born in the changing world.

5. Acknowledgements

I am grateful for my supervisors Anna-Liisa Laine and Fletcher Halliday for providing me with guidance during the whole process and giving me the opportunity to complete my thesis as part of such an interesting research project. I thank the whole field team, Ilmari Kohonen, Viviana Loaiza, Krista Raveala and Mikko Tiusanen, for help in the field work; all members of the Laine lab for support throughout the last year; Reto Nyffeler, Marc Mächler and Henry Väre for help with plant identification; and University of Helsinki and University of Zürich for funding the project.

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7. Appendices

Appendix 1 Table of the research sites. Coordinates (as WGS) and elevation (as meters above sea level) are reported from the 'main corner' of each site. Offset 1 = offset perpendicular to river Rhine. Offset 2 = offset parallel to river Rhine.

Site	Meadow	Elevation	Shape	WGS84.N	WGS84.E	Offset1	Offset2
I1	Im Bofel	766.3m	50m x 50m	46.871515	9.512329	4.94m	2m
I2	Im Bofel	737.4m	50m x 50m	46.871772	9.513894	2.74m	3.19m
I3	Im Bofel	711.5m	100m x 100m	46.867397	9.51093	1.8m	7.25m
I4	Im Bofel	702.4m	50m x 50m	46.868427	9.512794	2.13m	5.04m
I5	Im Bofel	684.5m	50m x 50m	46.869759	9.513897	4.29m	1.62m
I6	Im Bofel	702.2m	50m x 50m	46.874319	9.517722	5.2m	4.18m
I7	Im Bofel	648.5m	50m x 50m	46.874214	9.519005	2.03m	4.63m
A1	Arella	1020.6m	50m x 50m	46.873877	9.508583	3.86m	4.79m
A2	Arella	984.9m	50 m x 50m	46.871415	9.506058	3.13m	5.03m
A3	Arella	949.8m	50m x 50m	46.87088	9.507291	6.47m	6.76m
A4	Arella	1002.8m	50m x 50m	46.868932	9.502052	5.95m	4.55m
A5	Arella	1001m	50m x 50m	46.866705	9.499144	7.51m	3.9m
A6	Arella	981.9m	50m x 50m	46.864430	9.497368	2.03m	4.33m
N1	Nesselboden	1390.3m	50m x 50m	46.867567	9.486815	4.6m	7.2m
N2	Nesselboden	1405.5m	50m x 50m	46.86855	9.487988	3.18m	5.7m
N3	Nesselboden	1407.7m	50m x 50m	46.868775	9.488871	4.73m	5.73m
N4	Nesselboden	1420m	50m x 50m	46.8698	9.489261	3.7m	7.5m
N5	Nesselboden	1398.6m	50m x 50m	46.869485	9.490429	2.89m	5.09m
O3	Oberberg – Under Alp	1612.7m	50m x 50m	46.877719	9.494482	2.68m	5.19m
O4	Oberberg – Under Alp	1576.2m	50m x 50m	46.877238	9.495188	4.92m	5.29m
U1	Oberberg – Under Alp	1745.8m	50m x 50m	46.880264	9.492043	6.4m	4m
U2	Oberberg – Under Alp	1749.2m	50m x 50m	46.879824	9.491029	5.9m	5.4m

Appendix 2 Timing of grazing, vegetation and disease surveys and temperature measurement of each site. *Temperature data logger was moved temporarily off the field for 12 days 30.8.–10.9.2019 because of grazing. **The number of days the site had to recover after grazing, before the vegetation survey was carried out.

Site	Meadow	Elevation	Date of vegetation survey	Recovery days**	Date of the disease survey	Date of the temperature measurements
I1	Im Bofel	766.3m	10.7.2019	24	15.8.2019	7.8.–9.9.2019
I2	Im Bofel	737.4m	4.7.2019	18	13.–15.8.2019	7.8.–9.9.2019
I3	Im Bofel	711.5m	3.–4.7.2019	17–18	29.7.2019	7.–28.8.2019
I4	Im Bofel	702.4m	2.–3.7.2019	16–17	29.–30.7.2019	7.–28.8.2019
I5	Im Bofel	684.5m	27.6.2019–2.7.2019	11–16	18.–30.7.2019	7.–28.8.2019
I6	Im Bofel	702.2m	28.6.2019	12	13.8.2019	7.8.–11.9.2019
I7	Im Bofel	648.5m	5.–9.7.2019	19–23	30.7.2019	7.8.–11.9.2019
A1	Arella	1020.6m	16.–17.7.2019	17–18	8.8.2019	7.8.–12.9.2019
A2	Arella	984.9m	16.7.2019	17	15.–19.8.2019	7.8.–12.9.2019
A3	Arella	949.8m	15.–16.7.2019	16–17	8.8.2019	7.8.–11.9.2019
A4	Arella	1002.8m	12.7.2019	13	22.8.2019	7.8.–11.9.2019
A5	Arella	1001m	11.7.2019	12	7.8.2019	7.8.–11.9.2019
A6	Arella	981.9m	10.–11.7.2019	11–12	7.8.2019	7.8.–11.9.2019
N1	Nesselboden	1390.3m	23.7.2019	20	14.8.2019	7.8.–12.9.2019
N2	Nesselboden	1405.5m	22.–23.7.2019	19–20	2.8.2019	7.8.–12.9.2019*
N3	Nesselboden	1407.7m	19.–22.7.2019	16–19	2.–6.8.2019	7.8.–12.9.2019*
N4	Nesselboden	1420m	19.7.2019	16	6.–14.8.2019	7.8.–12.9.2019*
N5	Nesselboden	1398.6m	17.–19.7.2019	14–16	19.8.2019	7.8.–12.9.2019*
O3	Oberberg – Under Alp	1612.7m	24.7.2019	5	31.7.2019	7.8.–8.9.2019
O4	Oberberg – Under Alp	1576.2m	23.–24.7.2019	4–5	31.7.2019	7.8.–9.9.2019
U1	Oberberg – Under Alp	1745.8m	25.7.–1.8.2019	6–13	16.8.2019	7.8.–8.9.2019
U2	Oberberg – Under Alp	1749.2m	26.7.–1.8.2019	7–13	16.8.2019	7.8.–8.9.2019

Appendix 3 List of the plant taxa observed in the vegetation survey. Some plants were not identified but named with an ID code and this name was used throughout the survey.

1. *Achillea millefolium*
2. *Acinos alpinus*
3. *Aegopodium podagraria*
4. *Agrimonia eupatoria*
5. *Agrostis capillaris*
6. *Agrostis capillaris / A. schraderiana*
7. *Agrostis schraderiana*
8. *Agrostis schraderiana / A. stolonifera*
9. *Agrostis sp*
10. *Agrostis stolonifera*
11. *Ajuga reptans*
12. *Alchemilla sp*
13. *Alchemilla sp 1*
14. *Alchemilla sp 2*
15. *Anthericum ramosum*
16. *Anthyllis vulneraria*
17. *Arabis ciliata*
18. *Arabis sp / Arabidopsis sp*
19. *Arenaria serpyllifolia*
20. *Asperula cynanchica*
21. *Aster amellus*
22. *Aster bellidiastrum*
23. *Asteraceae sp*
24. *Bellis perennis*
25. *Brachypodium pinnatum*
26. *Briza media*
27. *Bromus erectus*
28. *Bromus hordeacus*
29. *Bromus inermis*
30. *Bromus sp*
31. *Bromus sp / Koeleria sp*
32. *Buphthalmum salicifolium*
33. *Campanula glomerata*
34. *Campanula rotundifolia*
35. *Campanula schleuzerii / C. rotundifolia*
36. *Campanula sp*
37. *Carduus defloratus*
38. *Carduus sp*
39. *Carex flacca*
40. *Carex sempervirens*
41. *Carex sp*
42. *Carex sp 1*
43. *Carex sp 2*
44. *Carex sp 3*
45. *Carex sp 4*
46. *Carex sp U2.3.1*
47. *Carex sp U2.8.2*
48. *Carlina acaulis*
49. *Carlina sp / Cirsium sp*
50. *Carlina vulgaris*
51. *Carum carvi*
52. *Centaurea jacea*
53. *Centaurea scabiosa*
54. *Cerastium fontanum*
55. *Cerastium sp*
56. *Cirsium acaule*
57. *Cirsium arvense*
58. *Clinopodium vulgare*
59. *Colchicum autumnalis*
60. *Crepis sp*
61. *Crepis sp / Leontodon sp*
62. *Cynosurus cristatus*
63. *Dactylis glomerata*

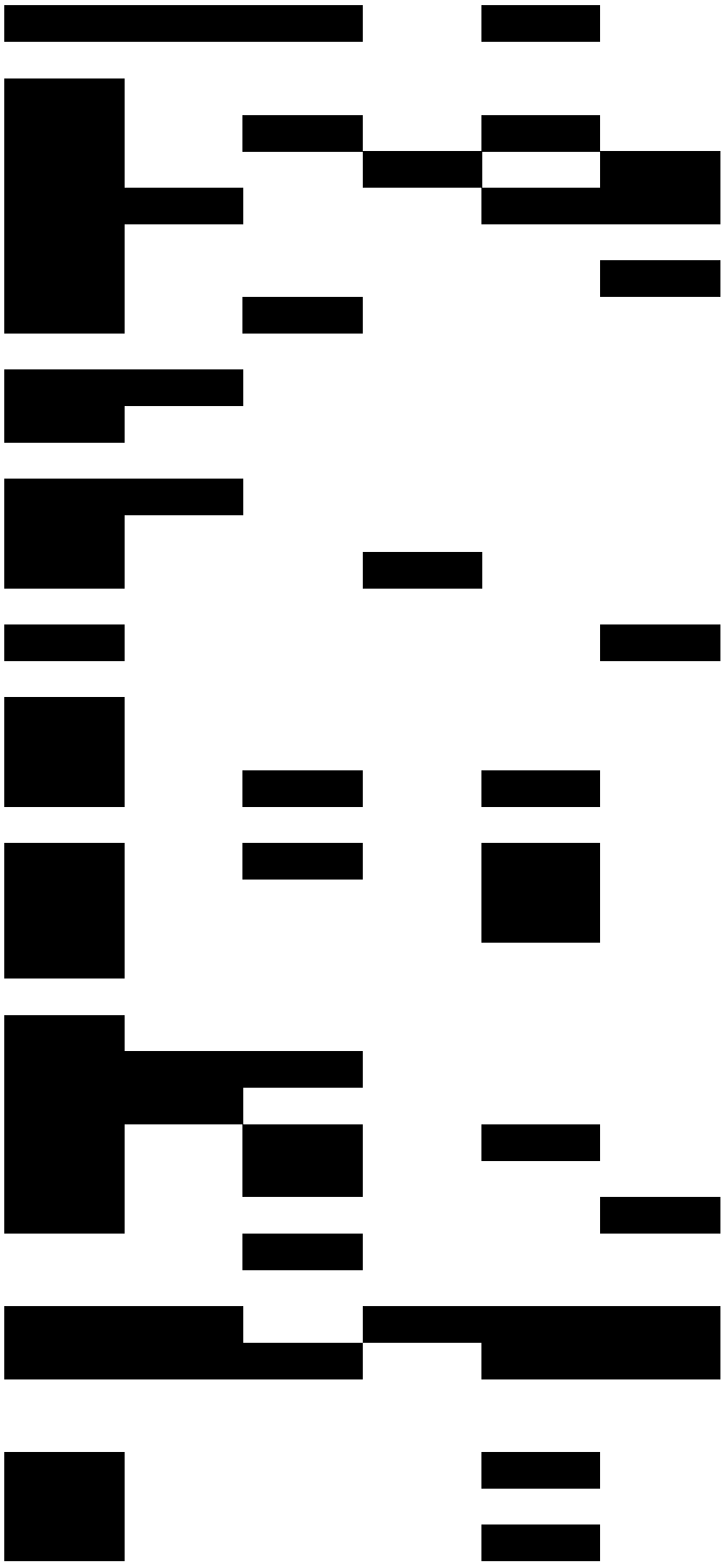
64. *Danthonia decumbens*
65. *Daucus carota*
66. *Daucus carota / Carum carvi*
67. *Deschampsia cespitosa*
68. *Dicot sp*
69. *Dicot sp 1 N1.5.2*
70. *Dicot sp 2*
71. *Dicot sp 2 N1.5.2*
72. *Dicot sp 5*
73. *Dicot sp N4.1.1*
74. *Dicot sp U2.3.3*
75. *Echium vulgare*
76. *Erica carnea*
77. *Euphorbia cyparissias*
78. *Euphrasia sp*
79. *Festuca ovina*
80. *Festuca rubra*
81. *Fragaria vesca*
82. *Galium sp*
83. *Galium verum*
84. *Gentiana sp*
85. *Geranium columbina*
86. *Geranium sylvaticum*
87. *Globularia sp*
88. *Helianthemum nummularium*
89. *Hepatica nobilis*
90. *Hieracium lactucella*
91. *Hieracium pilosella / H. hoppeanum*
92. *Hieracium piloselloides*
93. *Hieracium sp*
94. *Hippocrepis comosa*
95. *Homogyne alpina*
96. *Hypericum perforatum*
97. *Hypericum sp*
98. *Juniperus communis*
99. *Knautia arvensis*
100. *Larix europaeus*
101. *Lathyrus pratensis*
102. *Leontodon autumnalis*
103. *Leontodon hispidus*
104. *Leontodon sp*
105. *Leucanthemum vulgare*
106. *Linum catharticum*
107. *Lolium perenne*
108. *Lotus corniculatus*
109. *Lotus maritimus*
110. *Luzula sp*
111. *Maianthemum bifolium*
112. *Medicago falcata*
113. *Medicago lupulina*
114. *Molinia caerulea*
115. *Ononis spinosa*
116. *Orchidaceae sp*
117. *Orchis ustulata*
118. *Pastinaca sativa*
119. *Peucedanum oreoselinum*
120. *Phleum pratense*
121. *Pimpinella saxifraga*
122. *Pinus sp*
123. *Plantago atrata*
124. *Plantago lanceolata*
125. *Plantago major*
126. *Plantago media*
127. *Poa annua*
128. *Poa badensis*
129. *Poa pratensis*
130. *Poa sp*
131. *Poa sp A1.6.1*

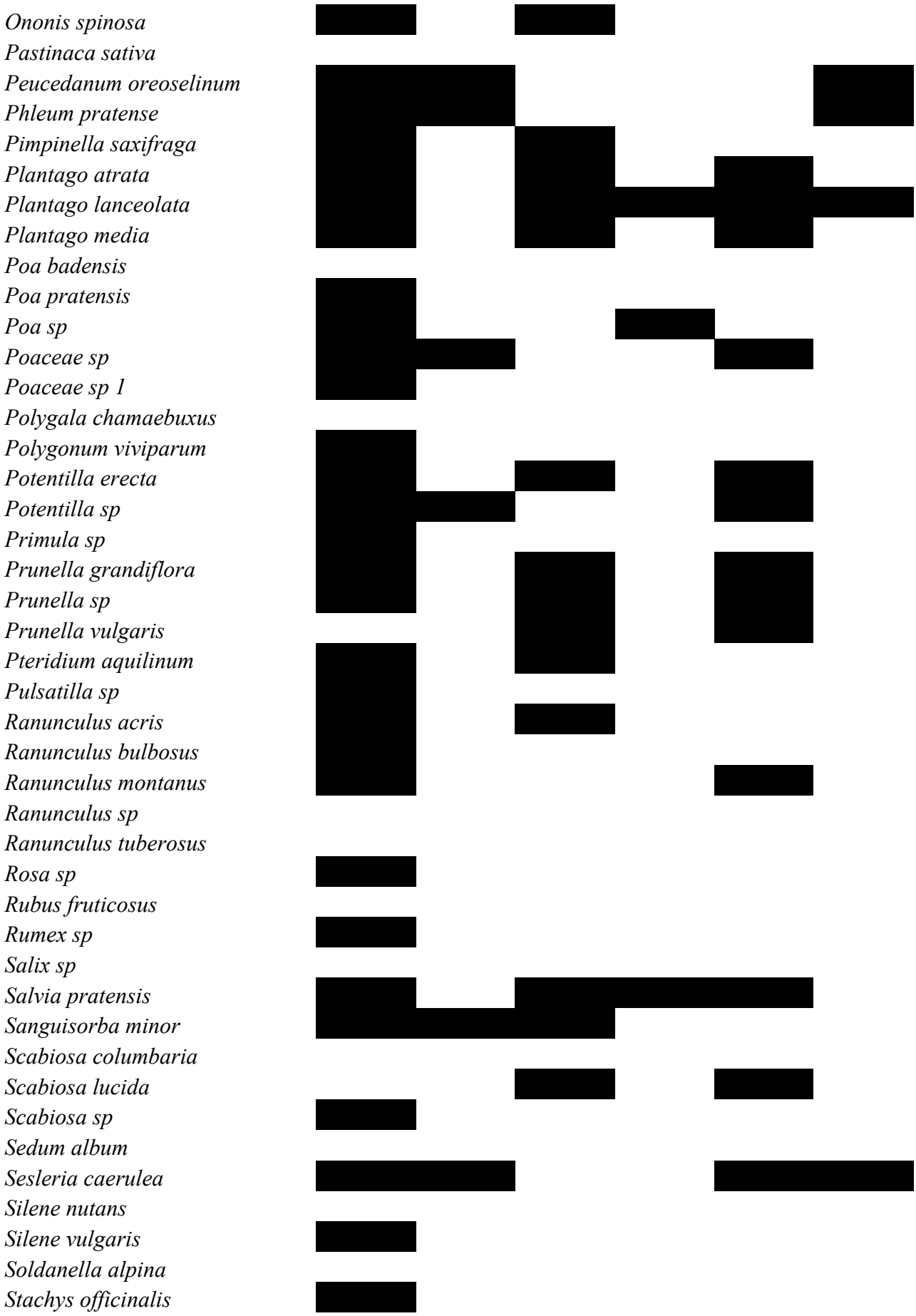
132. *Poaceae sp*
133. *Poaceae sp 1*
134. *Poaceae sp 2*
135. *Polygala alpestris*
136. *Polygala chamaebuxus*
137. *Polygala comosa*
138. *Polygala vulgaris*
139. *Polygonum viviparum*
140. *Potentilla erecta*
141. *Potentilla sp*
142. *Potentilla sp I7.1.1*
143. *Primula sp*
144. *Primula veris*
145. *Prunella grandiflora*
146. *Prunella sp*
147. *Prunella vulgaris*
148. *Pteridium aquilinum*
149. *Pulsatilla sp*
150. *Ranunculus acris*
151. *Ranunculus bulbosus*
152. *Ranunculus montanus*
153. *Ranunculus sp*
154. *Ranunculus tuberosus*
155. *Rosa sp*
156. *Rubus fruticosus*
157. *Rumex sp*
158. *Salix sp*
159. *Salvia pratensis*
160. *Sanguisorba minor*
161. *Scabiosa columbaria*
162. *Scabiosa lucida*
163. *Scabiosa sp*
164. *Sedum album*
165. *Sesleria caerulea*
166. *Silene nutans*
167. *Silene vulgaris*
168. *Soldanella alpina*
169. *Stachys officinalis*
170. *Taraxacum sp*
171. *Taraxacum sp / Leontodon sp*
172. *Teucrium chamaedrys*
173. *Teucrium montanum*
174. *Thymus serpyllum*
175. *Tragopogon pratensis*
176. *Trifolium montanum*
177. *Trifolium pratense*
178. *Trifolium repens*
179. *Trollius europaeus*
180. *Tussilago farfara*
181. *Vaccinium vitis-idae*
182. *Veratrum album*
183. *Veronica chamaedrys*
184. *Veronica officinalis*
185. *Veronica sp*
186. *Veronica spicata*
187. *Vicia cracca*
188. *Vicia sepium*
189. *Viola sp*

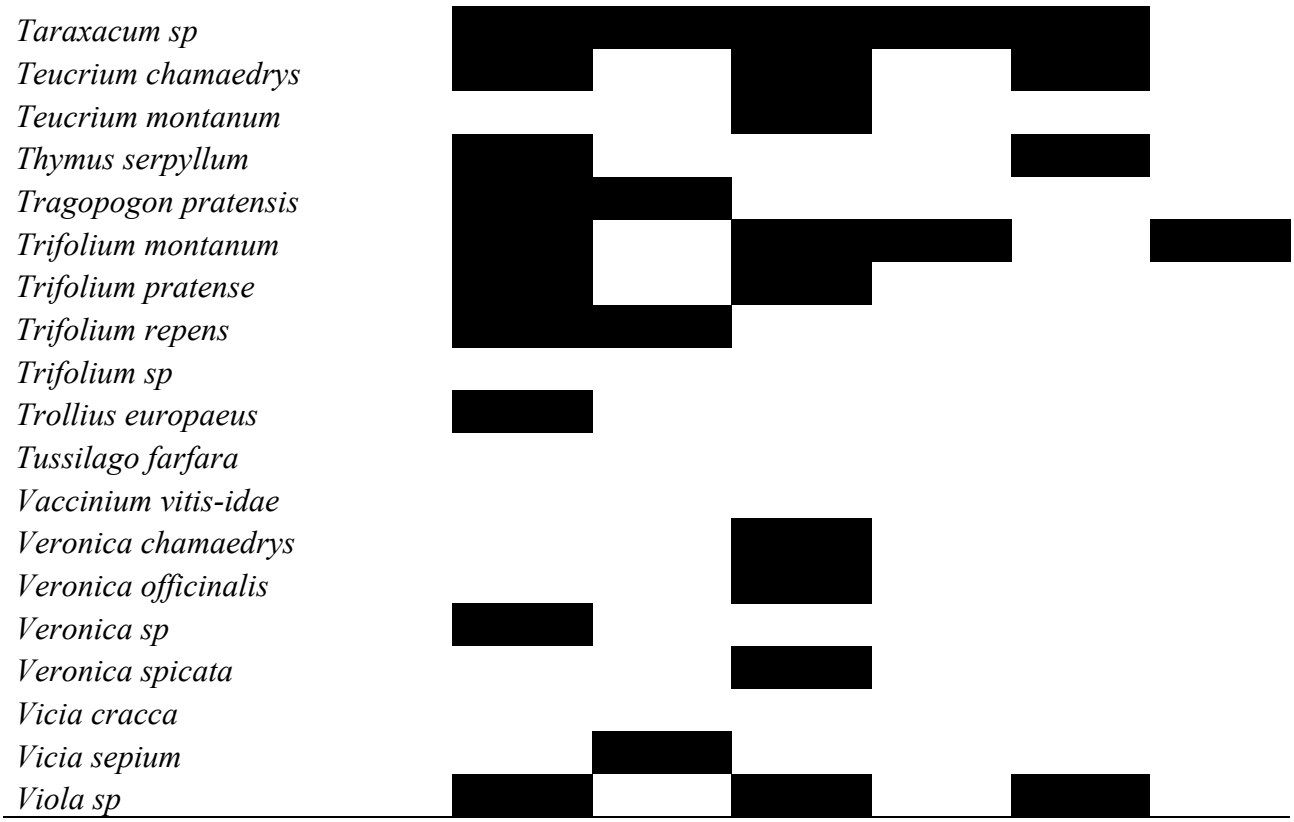
Appendix 4 Table of the plant species observed in the disease survey with the symptoms observed on each species. Blackened cells indicate that the symptom was observed on the species. The category 'Other fungal' includes unidentified fungal symptoms. The category 'Other' includes chlorotic and necrotic spots, leaf wetting, leaf curling and leaf choking.

Species	Leaf spot	Rust	Blight	Powdery mildew	Other fungal	Other
Achillea millefolium	■		■		■	■
Acinos alpinus	■				■	
Aegopodium podagraria	■	■				
Agrimonia eupatoria	■		■			
Agrostis capillaris	■		■			
Agrostis schraderiana	■					
Agrostis sp	■	■			■	
Ajuga reptans	■					
Alchemilla sp 1	■	■	■			
Alchemilla sp 2	■	■	■	■	■	■
Anthericum ramosum	■				■	
Anthyllis vulneraria						
Arabis ciliata						
Arenaria serpyllifolia						
Asperula cynanchica	■		■			
Aster amellus						■
Brachypodium pinnatum	■	■		■	■	■
Bromus sp						
Bromus sp / Koeleria sp	■	■		■	■	■
Buphthalmum salicifolium			■			
Campanula glomerata	■					
Campanula rotundifolia						
Carduus defloratus	■				■	
Carduus sp						
Carex flacca	■	■	■		■	
Carex sempervirens	■	■				
Carex sp	■					
Carex sp 1	■				■	■
Carex sp 2	■	■	■			■
Carex sp 3	■					
Carex sp 4	■					
Carex sp U2.3.1	■					
Carlina acaulis	■		■		■	
Carum carvi	■				■	
Centaurea jacea	■		■	■	■	
Centaurea scabiosa	■					

Cerastium fontanum
Cirsium acaule
Cirsium arvense
Clinopodium vulgare
Crepis sp / Leontodon sp
Cynosurus cristatus
Dactylis glomerata
Danthonia decumbens
Daucus carota
Echium vulgare
Erica carnea
Euphorbia cyparissias
Euphrasia sp
Festuca ovina
Festuca rubra
Festuca sp
Fragaria vesca
Galium sp
Galium verum
Gentiana sp
Geranium sylvaticum
Globularia sp
Helianthemum nummularium
Hepatica nobilis
Hieracium lactucella
Hieracium pilosella / H. hoppeanum
Hippocrepis comosa
Homogyne alpina
Hypericum perforatum
Knautia arvensis
Lathyrus pratensis
Leontodon autumnalis
Leontodon hispidus
Leontodon sp
Leucanthemum vulgare
Linum catharticum
Lolium perenne
Lotus corniculatus
Lotus maritimus
Luzula sp
Medicago falcata
Medicago lupulina
Molinia caerulea




Ononis spinosa
Pastinaca sativa
Peucedanum oreoselinum
Phleum pratense
Pimpinella saxifraga
Plantago atrata
Plantago lanceolata
Plantago media
Poa badensis
Poa pratensis
Poa sp
Poaceae sp
Poaceae sp 1
Polygala chamaebuxus
Polygonum viviparum
Potentilla erecta
Potentilla sp
Primula sp
Prunella grandiflora
Prunella sp
Prunella vulgaris
Pteridium aquilinum
Pulsatilla sp
Ranunculus acris
Ranunculus bulbosus
Ranunculus montanus
Ranunculus sp
Ranunculus tuberosus
Rosa sp
Rubus fruticosus
Rumex sp
Salix sp
Salvia pratensis
Sanguisorba minor
Scabiosa columbaria
Scabiosa lucida
Scabiosa sp
Sedum album
Sesleria caerulea
Silene nutans
Silene vulgaris
Soldanella alpina
Stachys officinalis


Taraxacum sp
Teucrium chamaedrys
Teucrium montanum
Thymus serpyllum
Tragopogon pratensis
Trifolium montanum
Trifolium pratense
Trifolium repens
Trifolium sp
Trollius europaeus
Tussilago farfara
Vaccinium vitis-idae
Veronica chamaedrys
Veronica officinalis
Veronica sp
Veronica spicata
Vicia cracca
Vicia sepium
Viola sp